JAN 16 1930

Registered at the G.P.O., Sydney, for Transmission by Post as a Newspaper. Published Weekly. Price 1s.

THE MEDICAL JOURNAL OF AUSTRALIA

Medical Lib.

VOL. II.—16TH YEAR. SYDNEY, SATURDAY, DECEMBER 14, 1929. No. 24.

'BYNIN' AMARA

Trade Mark

A POTENT TONIC

'BYNIN' AMARA is of special value in neurasthenia, particularly when associated with low blood pressure, anaemia and atonic dyspepsia; in convalescence it gives that impetus which often enables the system to overcome the aftermath of disease and to recover completely.

COMPOSITION.

Quinine Phosphate	1½ gr.
Iron Phosphate	2 ,,
Nux Vomica Alkaloids *equal to Strychnine*	½ ,,
'Bynin' Liquid Malt	1 oz.

DOSE: 2 to 4 fluid drachms in water twice or thrice daily after meals.

Further particulars and free sample will be sent on request.

ALLEN & HANBURYS (AUSTRALASIA) LTD.

13 MARKET STREET - - - - - SYDNEY, N.S.W.

Men can choose Gifts for Ladies at PEAPES

So that men can select in the atmosphere to which they are accustomed, gifts of quality and distinction for ladies, PEAPES now display many beautiful and rare articles eminently suitable for presentation to ladies.

21. Swan Presentation Pen and Pencil Sets for Ladies, 45/-, 52/6, 63/-, 72/6 and 77/6.

22. Ladies' Manicure Sets, 37/6, 42/-, 45/-, 50/-, 55/-, 63/-, 70/-, 84/-, 105/-, 142/- and 210/- per set

23. Houbigant's, Coty's, Caron's Perfumes and Eau de Colognes. All perfumes.

24. Ladies' Handbags, Envelope or Pouch Shape. Each, 27/6, 35/-, 42/-, 50/-, 55/-, 60/-, 70/-, 75/-, 84/-, 105/-, 115/-, 126/- and 147/-.

25. Ladies' Pure Irish Linen Handkerchiefs in folders of six. Embroidered corners. Prices, 14/6, 16/6, 18/6, 21/-, 25/-, 30/- and 35/-.

26. Ladies' Pure Irish Linen Handkerchiefs in folders of six. Lace corners and edges. Prices, 17/6, 22/6, 27/6, 35/- and 42/-.

If desired, gifts will be posted to any address with donor's card enclosed.

PEAPES

clothe men *AND* their sons

GEORGE STREET, *(Opposite Hunter Street.)*

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—16TH YEAR. SYDNEY, SATURDAY, DECEMBER 14, 1929. No. 24.

Authors of articles submitted for publication are requested to read the following instructions and to comply with them.

All articles must be typed with double or treble spacing. Carbon copies should not be sent. Abbreviations should be avoided, especially those of a technical character at times employed in ward notes. Words and sentences should not be underlined or typed in capitals. The selection of the correct type is undertaken by the Editors. When illustrations are required, good photographic prints on glossy gaslight papers should be submitted. Each print should be enclosed in a sheet of paper. On this sheet of paper the number of the figure and the legend to appear below the print should be typed or legibly written. On no account should any mark be made on the back of the photographic print. If no good print is available, negatives may be submitted. Line drawings, graphs, charts and the like should be drawn on thick, white paper in India ink by a person accustomed to draw for reproduction. The drawings should be large and boldly executed and all figures, lettering and symbols should be of sufficient strength and size to remain clear after reduction. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available. The reproduction of all illustrations, but especially of skiagrams, entails the sacrifice of time and energy and is expensive. Authors are expected to take a corresponding amount of trouble on the preparation of their illustrations, whether skiagrams, photographs, wash drawings or line drawings. The references to articles and books quoted must be accurate and should be compiled according to the following scheme. The order should correspond to the order of appearance in the article. The initials and surnames of the authors, the full title of the article or book, the full (unabbreviated) title of the journal in which the article appears, the date of the issue (day, month and year) and the number of the first page should be given in this sequence.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.
"The Treatment of Acute Poliomyelitis by Means of Human Immune Serum," by JEAN MACNAMARA, M.D. .. 838
"The Activity of Stored Antipoliomyelitic Serum in Experimental Poliomyelitis," by F. M. BURNET, M.D., Ph.D., and JEAN MACNAMARA, M.D. .. 851

REVIEWS—
Treatment by Physical Means .. 856
Surface Anatomy .. 856
The Evolution of Public Health Nursing .. 856
Incompatibility in Prescribing .. 856

LEADING ARTICLES—
Going Back to School .. 857

CURRENT COMMENT—
Poliomyelitis .. 858
The Use of Glycerin in Cæsarean Section .. 859

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—
Morbid Anatomy .. 860
Morphology .. 861

BRITISH MEDICAL ASSOCIATION NEWS—
Scientific .. 862
Nominations and Elections .. 864

MEDICAL SOCIETIES—
The Brisbane Hospital Clinical Society .. 864
The Medical Sciences Club of South Australia .. 866

CONGRESS NOTES— PAGE.
International Physiological Congress .. 867

NAVAL, MILITARY AND AIR FORCE—
Appointments .. 868

UNIVERSITY INTELLIGENCE—
The University of Sydney .. 869

SPECIAL CORRESPONDENCE—
Canada Letter .. 869

CORRESPONDENCE—
Tonsillectomy .. 870
Steinach's Operation .. 871
Duodenal Stenosis .. 871
Prolapsus Uteri .. 871
Tænia Saginata .. 871

TESTIMONIAL TO MR. W. FILMER .. 871

PROCEEDINGS OF THE AUSTRALIAN MEDICAL BOARDS—
New South Wales .. 872

BOOKS RECEIVED .. 872

DIARY FOR THE MONTH .. 872

MEDICAL APPOINTMENTS .. 872

MEDICAL APPOINTMENTS VACANT, ETC. .. 872

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 872

EDITORIAL NOTICES .. 872

THE TREATMENT OF ACUTE POLIOMYELITIS BY MEANS OF HUMAN IMMUNE SERUM.[1]

By JEAN MACNAMARA, M.D. (Melbourne),
Melbourne.

THE use of serum prepared from the blood of individuals who have passed through an attack of poliomyelitis has its basis on experimental work published in 1910 by Flexner and Lewis,[1] Landsteiner and Levaditi.[2] These workers demonstrated that the blood of such individuals possessed virucidal properties. In 1911 Netter,[3] in France, used human immune serum in the treatment of patients with acute forms of the disease. During the epidemic of 1916 in New York, Amoss and Chesney,[4] Draper[5] and others used convalescent serum. The method was revived again in New Zealand during 1924 and 1925, particularly by Dr. W. S. Robertson in Wellington.[6] At the Dunedin Congress in 1927, Dr. Hector[7] in a paper on the New Zealand epidemic quoted from the answers to a *questionnaire* sent to every part of the Dominion —a conclusion of the experience gained during the epidemic—"convalescent serum is a specific in promoting prompt recovery and in preventing paralysis if given in the pre-paralytic stage." In 1925 an epidemic of some 130 cases occurred in Victoria and because of the New Zealand results an attempt was made to prepare serum in Melbourne. The details of the initial activities have been published.[8][9] During the past two years several series of cases in which patients were treated in the preparalytic stage by means of human immune serum have been recorded. Of these the largest have been reported by Ayer, of Syracuse,[10] and by Aycock and Luther, of Boston.[11] Other smaller series have been recorded in France and in Canada. In Victoria since the Dunedin congress over 330 cases of poliomyelitis have occurred, 200 during 1928 and the majority of the remainder in 1929. I am indebted to the profession throughout the State for the opportunity given me since 1925 of following up the patients over long periods, of seeing early "suspect" cases and of treating any suitable patients with serum. The series of cases to be presented has occurred in Victoria in the period 1925 to 1929. In the earlier years lack of experience led to the administration of serum to a small number of patients who had reached the stage of stationary paralysis. In 1928 and 1929 the use of serum has been limited to persons with infections diagnosed as being in the preparalytic stage, to patients in the first twenty-four hours after the onset of paralysis with persistent fever and to those with older infections of the spreading type.

Classification of Patients.

In order to assess the results obtained by this therapy, the patients have been divided into several groups which will be considered separately: Group I, those who received serum in the preparalytic stage; Group II, those who received serum at the stage of early paralysis with persistent fever; Group III, those who received serum later than the first twenty-four hours after the advent of paralysis; Group IV, those with the ascending or spreading type. Groups III and IV will be considered before Groups I and II.

Group III.

Group III comprised twelve patients, eleven children and one adult, who received serum after paralysis had been observed for two or more days. Their histories are not reported in detail, but they have all been seen regularly and their progress noted and compared with that of others with similar initial paralysis among the control group of 123 non-serum treated patients whom it has been my privilege to observe over long periods. One in this group died some weeks later of pneumonia. The degree and rate of recovery of the muscle function and power in the other children have been equalled by others in the control series who have received equivalent after treatment. The results from administration of serum after paralysis has occurred for some days and is apparently stationary and the patient afebrile, do not justify the use of serum at this stage.

Group IV.

Group IV, the spreading, ascending type, comprises 22 patients. Three of these cases were recorded in 1927. Nineteen have occurred since. The ages of the patients vary from three to fifty-six, the greatest incidence occurring between the years

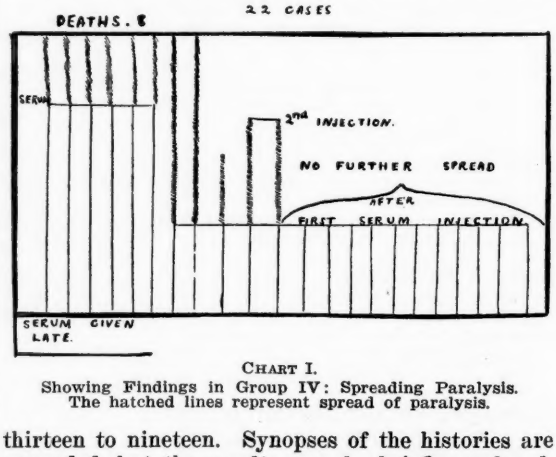


CHART I.
Showing Findings in Group IV: Spreading Paralysis.
The hatched lines represent spread of paralysis.

thirteen to nineteen. Synopses of the histories are appended, but the results may be briefly analysed. In ten instances no further paralysis occurred after the administration of serum. In two others paralysis extended between the first and second injection and did not extend after the second. In the remaining nine patients paralysis extended to other limbs and other muscles after serum had been given. In eight of these death occurred from spread to the bulbar centres. At the time of administration of

[1] Read at the Australasian Medical Congress (British Medical Association), Third Session, Sydney, September, 1929.

serum to six of those whose infections proved fatal, there was evidence of involvement of the respiratory centres—concentration on breathing, anxiety, loss of intercostal or of diaphragmatic movement. One child, Case XI in the series, received serum when *in extremis* six hours before death. In one case after an interval of quiescence of seven days the development of headache, rise in temperature, increase in the spine sign, hyperæsthesia in one arm led us to expect further paralysis. Serum was given and paralysis did not occur. Two other cases of the "jump" variety had occurred in the non-serum treated control series. In these, after two or three weeks of stationary paralysis with subsidence of symptoms, an extension occurred and in each instance death from respiratory failure.

In the three consecutive cases of 1925 and 1926 the administration of serum had been followed by the arrest of spreading paralysis. It was therefore decided to continue the use of serum in this type of infection when sufficient stocks were available, but on the results set forth in the table appended it must be admitted that there is no guarantee that the administration of serum will prevent further spread. Nor is this fact surprising when one remembers the microscopical pathology, the vessels choked by the aggregation of cells around their walls, the hæmorrhage, the œdema which must be in existence for some hours before paralysis is manifest clinically. For this reason it is problematical in any given case whether any drug or immune substance administered by blood stream or by the theca can reach the site of the lesion. The criticism may be offered that the treatment of this series was not sufficiently energetic or persistent, that injections should have been given intrathecally and repeated, but the interference with respiration manifested by many of the patients, as evidenced by anxiety or distress on manipulation or movement, seemed to contraindicate disturbance and in the majority it seemed wiser to endeavour to establish immunity by means of one large intravenous injection than by any intrathecal medication. In only one, Case VIII, was an attempt made to combine the administration of serum with the intravenous administration of hypertonic saline solution, as has been suggested by Aycock.[2]

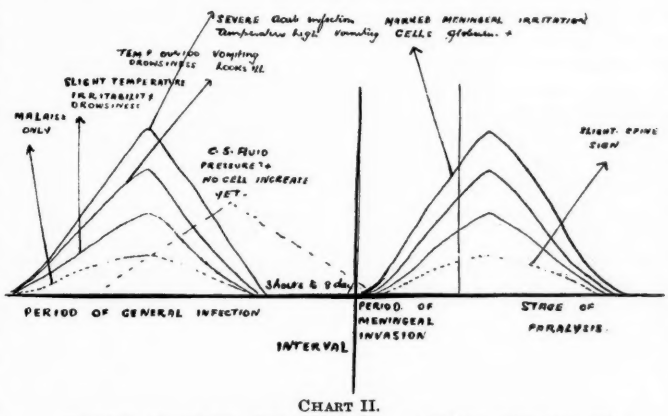


CHART II.
Showing Dromedary Type of Draper. Abortive infections end after the first hump.

Group I: The Preparalytic Stage.

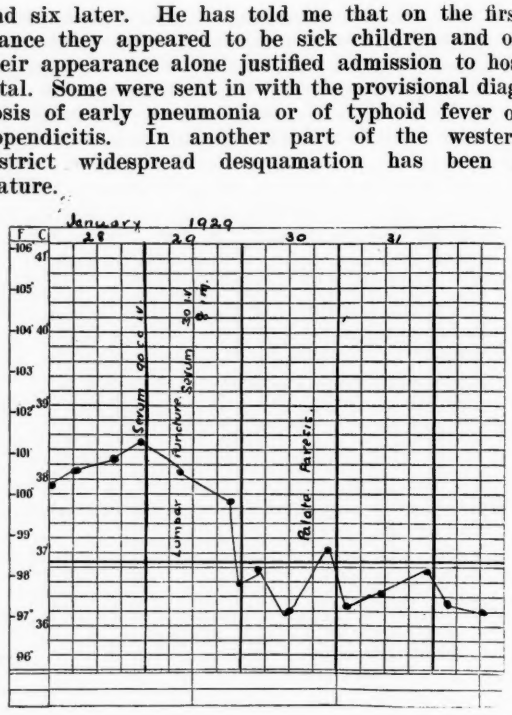
With a disease such as poliomyelitis, where the mortality and the crippling sequelæ result from lesions to the spinal cord cells, our endeavour should be to find a method of treatment which will abort the illness before damage has occurred to the cells. This fact emphasizes the importance of the recognition of the illness in its preparalytic stage. In the older epidemics of 1898 and 1908 a certain percentage of the cases had apparently a hyperacute or fulminating onset, the onset of the illness synchronizing or being followed very closely by the onset of paralysis. Fortunately this has not been the experience in the Victorian cases during the last few years. Of 216 patients whose detailed histories I have been given, there is only one whose infection may possibly be regarded as fulminating. In the others the onset of paralysis has been preceded by illness extending over several days with or without an intermission—the dromedary or the straggling type described by Draper. The "dromedary" type has been common. Of 80 patients admitted to the Children's Hospital, Melbourne, in the acute stage, only three sought admission within twenty-four hours of the onset of illness, eight within two days, twenty-two within two to four days, thirty within four to seven and and eighteen within seven to fourteen days from the onset.

The syndrome of the preparalytic stage can be recognized. It varies slightly in the different age groups, in babies under twelve months, in children and in adults, but the experience in Victoria has corroborated that of the New Zealand and American observers—that it is possible to diagnose poliomyelitis before paralysis has occurred. It is possible to be reasonably certain on the clinical examination before seeking the confirmation of examination of the cerebro-spinal fluid. In times of epidemic recognition is easier, the percentage of cases recognized early, higher. In the country centres where several cases have occurred, the resemblance of one case to another and the definite points of difference from other acute illnesses have impressed the physicians.

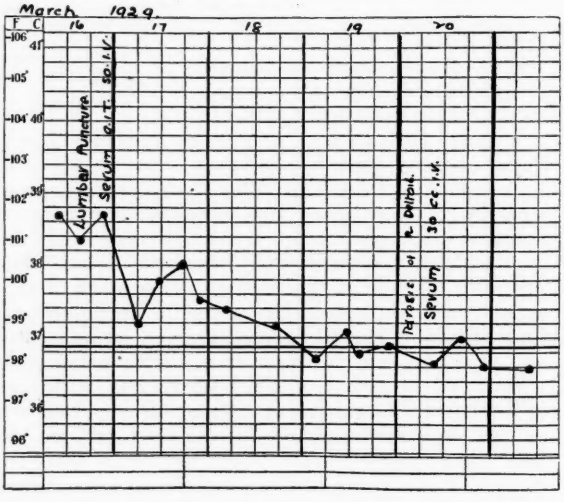
The symptoms may be briefly outlined as follows: Fever is usually present, rarely high and in the majority of adults has been no higher than 37·8° C. (100° F.). Headache is frequently complained of by older children and is sometimes exceedingly severe. Drowsiness when the child is left alone and irritability when it is disturbed is the common story of the parent; the irritability and drowsiness differ from the picture presented by a child with meningitis. If time be given to the examination of

the child and patience be used in making friends and reassuring him, the usual finding is one of cooperation up to a point. The child helps more in the examination than does the subject of tuberculous or cerebro-spinal meningitis. Pain in the neck is a frequent complaint; among the adults it has been agonizing. Sometimes pain is referred to a limb which may become hyperæsthetic. Vomiting once has been usual. In several young adults severely paralysed, persistent vomiting has been an early symptom. Convulsions have not been common and repeated convulsions exceedingly rare. Tremor of the hands, localized sweating, photophobia, retention of urine do not occur in every case, but any one of these symptoms is a valuable point. Constipation has been the rule. Clinical examination should be careful enough to exclude other conditions causing acute illness, such as tonsillitis, pyelitis, early pneumonia. The most helpful single sign in the differentiation from other acute illnesses has been the spine sign. By this I do not mean the head retraction or the degree of opisthotonus associated with tuberculous or cerebro-spinal meningitis. Occasionally it is sufficiently pronounced to resemble severe cerebro-spinal meningitis or tetanus. Case IX in the preparalytic series was such a one. But if we look for neck stiffness of the degree we see in cerebro-spinal meningitis, the majority of preparalytic poliomyelitis infections will be missed. The spine sign is best described as a voluntary disinclination to flex the spine anteriorly because it hurts. It is not wise to try to elicit it with the child in a recumbent position by putting the hand under the head and jerking it forward. Almost any febrile child will resent such handling. To determine whether the spine sign is present the child is held comfortably or nursed and asked to kiss his knee. One frequently sees him commence in good faith and give up the attempt because of pain and no bribing of a penny or even a shilling on the knee will persuade him to complete the flexion of the spine if this sign is present. A modification described as Amoss's sign is useful in older children and in adults. When placed in the sitting position on a firm table or chair, the position assumed is that of a tripod, the child conveying part of the weight of the body through the arms. If asked to put the hands on the head or to fold the arms, he begins and rapidly returns to the tripod position to ease the pain in the back. In babies one has frequently to rely on passive flexion to estimate whether pain is caused by the movement.

The degree of toxæmia has varied in different localities. Early in each season, in November and December, the infections have been mild in their initial stages, but in February, 1928, and in April, 1929, the fever has been higher. In different localities, even in minor details, the infections have resembled one another and differed in minor details from those in other districts. For example, at Geelong Dr. Langlands had a series of patients, six of whom were admitted in the preparalytic stage, and six later. He has told me that on the first glance they appeared to be sick children and on their appearance alone justified admission to hospital. Some were sent in with the provisional diagnosis of early pneumonia or of typhoid fever or appendicitis. In another part of the western district widespread desquamation has been a feature.



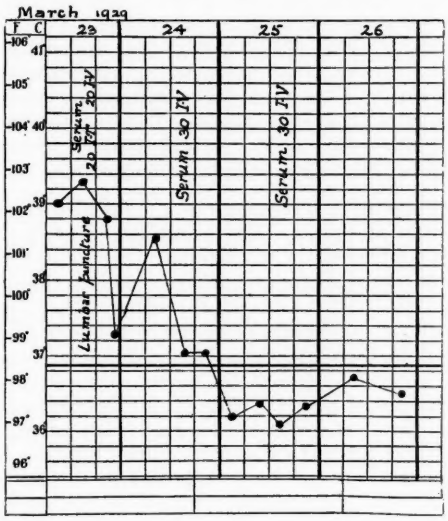
Temperature Chart, I, of female patient aged four years (Case XXIX). The patient was one of those in the preparalytic series. The spine sign was described as "++" and the cells numbered 65 per cubic millimetre. Paralysis of the palate occurred on the third day. Recovery was complete.



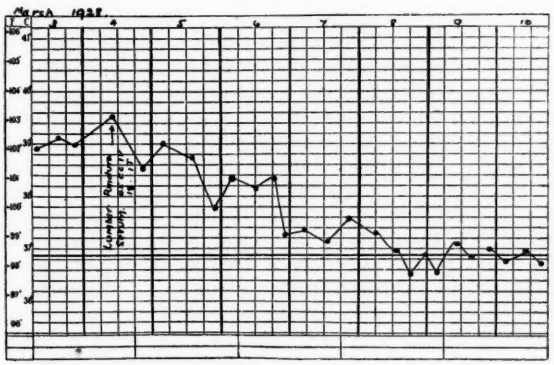
Temperature Chart, II, of male patient aged ten years (Case XXXIV). The patient was one of those in the preparalytic series. The spine sign was described as "+." The cerebro-spinal fluid contained 40 lymphocytes per cubic millimetre. The patient developed paresis of the right shoulder muscles in March, 1929, and recovery was complete in July.

The differential diagnosis of other suspect conditions covers a wide field of general medicine. In the four years during which I have had the opportunity of observing in consultation patients with

suspicious symptoms, there have been cases of tuberculous meningitis, cerebro-spinal meningitis, influenzal meningitis, post-vaccinal encephalitis (one), typhoid meningitis, osteomyelitis with toxæmia and few localizing signs, lead encephalopathy (four), injury to the limb, uræmia, *otitis media* with facial nerve paralysis, pneumonia with meningismus, syphilitic pseudo-paralysis, pyelitis, erythrœdema, scurvy, acute rheumatism.



Temperature Chart, III, of patient (Case XL). The patient was in the preparalytic stage. The spine sign was described as "++." The cells in the cerebro-spinal fluid numbered 150 per cubic millimetre. The patient developed paresis of the right facial muscles in March, 1929, with paresis of the right deltoid and the right *quadriceps* muscle. Recovery was complete by July.



Temperature Chart, IV, of female patient in the preparalytic series. The patient's brother, aged fourteen years, had died six days before from poliomyelitis. The cerebro-spinal fluid contained 331 cells per cubic millimetre. The globulin was described as "++" and the spine sign as "+++." No paralysis occurred.

If a condition is suspicious clinically of preparalytic poliomyelitis—and the more opportunity one has for seeing early cases, the more one appreciates the syndrome presented—then a lumbar puncture is justified. It is justified only if provision is made for immediate bedside examination of the cerebro-spinal fluid and if immune serum is available.

In 1925, when we were trying to place serum therapy for poliomyelitis on a satisfactory basis in Victoria, Dr. Reginald Webster, of Melbourne, warned us against advising promiscuous lumbar puncture in doubtful poliomyelitis. Dr. Webster[13] drew our attention to original work he had done in 1919 on laboratory animals given a pneumococcal septicæmia. In these animals when subjected to lumbar puncture the incidence of pneumococcal meningitis was greater than in those in which lumbar puncture was not performed. Subsequent work of the Rockefeller Institute workers[14] on the alteration of the meningo-chorioidal defence in poliomyelitis by lumbar puncture confirmed this warning. The clinical experience of the last three years gives further confirmation. In the series of preparalytic patients presented there are three, numbers VI, X and XXIII, and in the early paralytic group there are two, numbers IX and XIII, in whom after the withdrawal of a large amount of fluid by lumbar puncture an interval of twelve to thirty-six hours was allowed to elapse before serum

FIGURE I.
Showing paresis of the right shoulder muscles in Case XXXIII.

was administered. Of these one patient, number XX, died six hours after receiving serum. The histories of the other two preparalytic patients are as follows:

CASE VI: A boy of six years, after four days of illness with high fever, the temperature being 40·5° C. (105° F.) with photophobia, retention of urine, marked spine sign, was diagnosed clinically as suffering from preparalytic poliomyelitis and the diagnosis was confirmed by examination of the cerebro-spinal fluid, of which a large amount was withdrawn. An interval of fifteen hours was allowed to elapse before serum was given. His right leg became severely paralysed twelve hours after administration of the serum and twenty-seven after lumbar puncture.

CASE X: A girl of three was febrile, with a temperature of 38·3° C. (101° F.), had pains in the neck and back and retention of urine, hyperæsthesia and photophobia. Lumbar puncture was performed after thirty-eight hours of illness and again after sixty hours and an interval of eighteen hours was allowed to elapse between the second lumbar puncture and the administration of serum. The following day both legs were severely paralysed and have improved very little since.

Two patients in the early paralytic group give similar histories. These two patients, VI and X, are the only ones of the preparalytic series to be

severely paralysed. They illustrate the following points: (i) They were diagnosed clinically and by fluid examination as being in the preparalytic stage. Their history and appearance were identical with those found in many other cases occurring at the same time, proving once more that the preparalytic stage can be recognized. (ii) Lumbar puncture and withdrawal of large quantities of cerebro-spinal fluid should not be undertaken in a suspected case unless facilities are available for immediate examination of the fluid and unless immune serum is available.

To be of any value the examination for cells in the cerebro-spinal fluid must be carried out without delay. It has been shown by Lyon[15] that when the fluid is allowed to stand after withdrawal, cytolysis occurs in the early pleocytosis of acute poliomyelitis and that it is more active towards the multilobed elements than the mononuclear. This fact has been observed in this series when examination has been repeated by the same observer after the fluid has been allowed to stand. For this reason the procedure usually adopted with other meningeal infections, namely, obtaining fluid by lumbar puncture in a suspicious case, posting it to a distant laboratory and awaiting the results cannot be recommended with poliomyelitis. The actual withdrawal of fluid is dangerous unless immune serum is given forthwith and the examination after standing may be misleading. The procedure adopted in Melbourne and since the post-graduate course of 1928 in many country centres throughout the State has been as follows.

A careful clinical examination having failed to reveal any other cause for the illness, the consideration of the symptoms and signs leading one to the provisional diagnosis of preparalytic poliomyelitis, it is decided to obtain cerebro-spinal fluid by lumbar puncture. Before this is undertaken the microscope and counting chamber are set ready for use. A laboratory lamp is part of the equipment carried. The reagents for globulin examination are placed in readiness with the toluidin blue for staining. The needle is inserted, a few drops of fluid are withdrawn and the stillette reinserted and kept in position while the fluid is examined. If there is no increase in cells, the needle may be withdrawn and minimal damage has been done. If the cells are increased, it may be necessary to stain with the toluidin blue to discriminate mononuclear from polymorphonuclear cells.

If it is decided to give serum, it can then be given intrathecally and the patient thus saved a second lumbar puncture. In patients in whom the history has been at all suggestive of tuberculous meningitis, the chlorides of the cerebro-spinal fluid have been estimated. This test has been found of the greatest value. When care has been taken to take the fluid into a dry test tube, the chloride content in poliomyelitis has not been reduced. In tuberculous meningitis reduction occurs, the fall being progressive as the disease goes on. In two suspect cases the high chloride content of the fluid led to the estimation of the blood urea with the establishment of the diagnosis of uræmia. The findings of the examinations of the cerebro-spinal fluid of this series have been parallel to those in other countries. As the preparalytic stage progresses towards paralysis, so the proportion of the multilobed elements in the fluid falls and the mononuclear elements increase. Very high counts have been exceptional. Our figures are rather lower than the American figures and in the paralysed patients the severity of paralysis did not run parallel to the increase in cells. Globulin has been increased, but not greatly and frequently twenty minutes had to elapse before a reaction was obtained by means of the ammonium sulphate test. The examination of the cerebro-spinal fluid eliminates other meningeal infections resembling early poliomyelitis.

The apparatus required for administration of the serum is simple. The examination of the fluid having confirmed the provisional diagnosis of poliomyelitis, the stillette is withdrawn from the needle and as much fluid allowed to escape as is possible. The tubing and funnel containing serum slightly warmed is then attached to the lumbar puncture needle by a fitting and the serum allowed to flow in by means of gravity. The amount given has been slightly less than that of the fluid withdrawn. The dosage and method of administration in this series have varied, but we have followed in the majority of cases that adopted by Amoss and Chesney in America and Robertson in New Zealand—intrathecal injection at the onset, followed by a large intravenous injection and perhaps by a repeated intravenous injection on the following day. Other workers, such as Ayer and Netter, use repeated intrathecal injections, but we have had apparently larger stocks of serum available and could afford to give a larger intravenous dose. Shaw, Thelander and Fleischner[16] describe a small series of patients treated by intramuscular injections with satisfactory results. Theoretically, intravenous injections are preferable for the same reason that diphtheria antitoxin is given intravenously to a patient with laryngeal diphtheria, namely, that of urgency. The recognition of preparalytic poliomyelitis is an urgent problem, where time matters as much as in acute osteomyelitis or in a case of intussusception. In many points the aim of treatment in poliomyelitis and in intussusception is the same—to correct the condition before death of tissue has occurred through interference with the vascular supply. The dosage used has varied with individual cases. Rarely have less than sixty cubic centimetres been given. The amount can be read in the series attached. Before we had the monkeys to help us assess the potency of stale material, I deemed it wiser to use a relatively larger dose of serum prepared twelve months previously than of fresh material. As a rule the temperature falls at once. If a rise occurs during the next twenty-four hours, a second dose of serum has been given. No reaction has been observed in any patient, though looked for care-

fully. The intrathecal injection of twenty to twenty-five cubic centimetres of serum has not been followed by any evidence of increased meningeal irritation. Freedom from unpleasant reactions and rapid improvement in the general condition of the patient have been the rule and have justified the extra trouble involved in the preparation of a sterile serum free from any added antiseptic such as tricresol.

The patients treated in the preparalytic stage were patients whose doctors suspected the condition. In some cases the parents did. The findings in the fluid are given in the synopsis and the salient points in the history. Two cases have been discussed in detail—the two cases in which delay occurred between lumbar puncture and administration of serum. Of the remainder, very careful examination of muscles, where possible, by means of the spring-balance test of Lovett, has been carried out and thus very fine differences in muscle power detected. In several instances this careful examination disclosed paresis which had been missed by ordinary clinical examination, for example, Case II, "paresis of the left quadriceps," Case IX, "paresis of right *gluteus medius* for eight weeks." In sixteen instances these relative weaknesses were observed. This experience bears out that of other observers. In the older literature one reads of series of preparalytic patients treated by means of serum, escaping paralysis entirely. In the large series reported by Ayer[9] and Aycock,[10] particularly by the latter, where every child was examined by the trained workers of the Harvard Infantile Paralysis Commission, relative differences in power were discovered in children whom a more casual examination would have passed as having escaped paralysis entirely. Of the forty-two preparalytic patients of this series, one died six

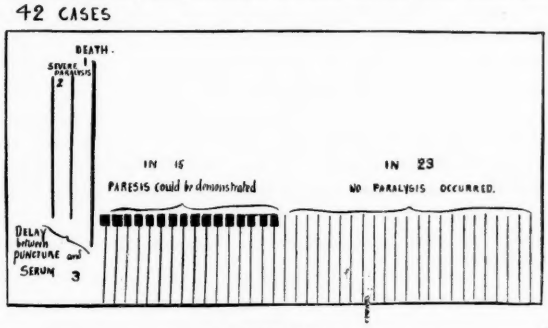


CHART III.
Showing Findings in Group I: Preparalytic Stage.

hours after serum was given. Two became severely paralysed—the two in whom delay occurred. Sixteen others had weakness for some weeks which could be demonstrated, but they will have no residual handicap therefrom. Twenty-three escaped without paralysis as far as could be detected on repeated examination. The sixteen in whom paresis could be demonstrated, have been useful in that, coming as they did at the same time as the others, with the same clinical picture, the same fluid findings, they served to convince even sceptics who saw them, that we were dealing with the one condition in all the cases.

FIGURE II.
Showing paresis of the right shoulder muscles in Cases XXXIII and XXXIV.

Group II.

The group of patients treated in the first twenty-four hours after the advent of paralysis comprised nineteen, all febrile at the time of administration of serum. Sixteen of these had no further paralysis. In one big girl extension to the lower limbs occurred two days later. In two others, in whom the same

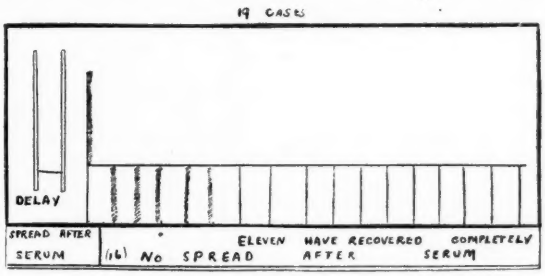


CHART IV.
Showing Findings in Group II. Serum was given at the stage of early paralysis.

delay between lumbar puncture with removal of a large amount of fluid and administration of serum occurred, paralysis extended. Of these nineteen patients eleven have made a complete recovery.

Discussion.

The difficulty in discussing poliomyelitis and the result of any method of therapy lies in the fact that in any given patient seen in the early stage, the prognosis varies between death on the one hand and complete recovery on the other, with any degree of paralysis as an intermediate possibility. The only accurate test of any method would be the treatment of alternate patients diagnosed before paralysis. This has not been done.

The epidemics of Victoria in 1928 and 1929 have not been mild. The mortality has been high (14·6%

during 1928), the severe infections frequent. In the latter months of 1928 a survey was made of the cases which had occurred during the year and a classification made according to the degree of residual paralysis. The same standards have been applied to the patients treated by serum in the pre-paralytic and early paralytic stage and while the one series cannot be regarded as a control series for the other, yet the comparison of the end results is instructive. They are shown in Table I. The figures are in percentages.

TABLE I.

Finding.	Patients Treated by Serum.		123 Patients Not Treated by Serum.
	Pre-paralytic Stage.	Early Paralytic Stage.	
Percentage mortality	2·3	—	21·6
Percentage with paralysis so severe that permanent invalidity is probable	2·3	—	9·6
Percentage with severe paralysis, yet probably capable of some locomotion ultimately	2·3	11	38·4
Mild paralysis, not severe enough to interfere with a self-supporting life (probably complete recovery)	9·5	31	24
Complete recovery at the date of the last examination	83·3	58	8

If we omit the three cases in the preparalytic group and the two others among the early paralytic series in which delay occurred between puncture and serum, the results read as shown in Table II.

TABLE II.

Finding.	Patients Treated by Serum.		123 Patients Not Treated by Serum.
	Pre-paralytic Stage.	Early Paralytic Stage.	
Mortality	—	—	21·6
Permanent incapacity	—	—	9·6
Severe paralysis compatible with some locomotion	—	—	38·4
Mild paralysis (probable complete recovery)..	15·3	35·3	24
Complete recovery at date of the last examination	84·7	64·7	8

These results are parallel with those reported by others, summed up by Aycock in his conclusion:

That the administration of human immune serum is beneficial, as evidenced by:

1. A low mortality rate,
2. A low average total paralysis and
3. A strikingly low proportion of paralysis of the severer grades.

Conclusion.

To conclude, the recognition of the preparalytic stage is essential for successful serum therapy.

Administration of serum within twenty-four hours after paralysis has occurred in a febrile patient is justified and is usually followed by fall of temperature and arrest of paralysis.

If given later, when paralysis is stationary, serum is of no value.

In the ascending group the administration is justified if stocks permit, but there is no guarantee that paralysis will be arrested.

Acknowledgements.

I acknowledge my indebtedness to the medical profession throughout Victoria for the cooperation during the last four years which which has made this work possible. The cases presented, the careful clinical records have been furnished by doctors in town and country. The series of cases represents the clinical report of a State-wide organization and while I have through the generosity of my colleagues had the opportunity of seeing these cases and examining the fluid, in many instances the work has been carried out by the patient's doctor. I would thank especially Dr. F. H. Langlands, of Geelong, and Dr. J. I. Connor, of Coleraine, for allowing me to include the reports of their large series of cases; the medical staffs of the Melbourne metropolitan and Geelong hospitals for the facilities given me, and the honorary and resident medical staff of the Children's Hospital, Melbourne, for advice and encouragement, particularly to Dr. Stewart Ferguson and Dr. Lionel Hood.

To Dr. Morgan, of the Commonwealth Serum Laboratories, and Dr. Kellaway and Dr. Burnet, of the Walter and Eliza Hall Institute, I am indebted for continued help and collaboration.

I would take this opportunity of thanking the Committee of the H. V. McKay Charitable Trust for the guarantee of finance for serum for country patients.

To the members of the Poliomyelitis Committee of Victoria, Dr. J. Dale, Dr. R. H. Fetherston, Dr. H. N. Featonby, Dr. P. A. Stephens, Dr. W. S. Summons and Dr. P. J. Flanagan, who have controlled the civic and financial side of the work since its inception in 1925, I am indebted for permission to publish these records. Finally, I acknowledge my great indebtedness to Mr. Kent Hughes whose initiative in 1925 gave me the opportunity to undertake this work.

The patients were under the care of the following practitioners:

Group I.

Dr. Birnie, Case 37.
Dr. Campbell, Case 22.
Dr. Connor, Cases 15, 16, 17, 18, 24.
Dr. Crawcour, Case 5.
Dr. J. C. Craig, Case 24.
Dr. Dinwoodie, Case 11.
Dr. Frayne, Case 4.
Dr. Graham, Case 30.
Dr. Hallam, Case 12.
Dr. Hamilton, Case 26.
Dr. Heslop, Case 42.
Dr. Langlands, Cases 31, 32, 33, 34, 35, 36.
Dr. Littlejohn, Cases 19, 30.
Dr. Macnamara, Case 38.
Dr. Mitchell, Case 6.
Dr. Norris, Cases 12, 13, 37.
Dr. Pern, Case 9.
Dr. Piper, Case 7.
Dr. Rennick, Case 10.
Dr. Rosenove, Case 1.
Dr. Seeley, Cases 20, 21.
Dr. Spencer, Cases 8, 25.
Dr. Summons, Case 23.
Dr. A. J. Woods, Case 14.
Children's Hospital (Dr. Ferguson, Dr. Forsyth, Dr. Hood, Dr. Stephens, Dr. Upjohn), Cases 2, 3, 27, 28, 29, 39, 40 and 41.

SYNOPSIS OF CASES TREATED BY HUMAN IMMUNE SERUM.

GROUP I.—PREPARALYTIC.

Group Number.	Date.	Age.	Duration of Illness in Days.	Temperature.	Spine Sign.	Headache.	Other Symptoms.	Cerebro-Spinal Fluid Findings.				Serum in c.cm.[4]		Result.	Comments.
								Number of Cells.	Type of Cells[3] or Description of Fluid.[2]	Globulin.	Chlorides in mgm. per 100 c.cm.	First Dose.	Second Dose.		
1	13.3.26	5	½	37·8° C. (100° F.)	++	+	Pains in legs and behind knees.	200	Lm.	–		20 I.T. 20 I.V.	12 I.V.	No paralysis.	
2	27.11.27	4	¾	37·8° C. (100° F.)	+	+	Pains in legs, paralysis right sixth nerve.	30	Lm.	–		60 I.V.		Paresis left quadriceps.	Recovery perfect in four months.
3	4.1.28	3	3	37·2° C. (99° F.)	+++	–	Drowsy, irritable.		B.S., I.P.	–		40 I.M.		No paralysis.	
4	6.1.28	4	1	37·2° C. (99° F.)	++	–	Drowsy, irritable.	35	70% Lm. 30% Pm.	–		10 I.V. 20 I.M.		No paralysis.	His brother had right arm paralysed four days before.
5	29.1.28	4	⅛	37·4° C. (90·4° F.)	+	–	Drowsy, pain in left leg.	280	80% Lm.	+		10 I.T. 40 I.M.		Paresis of left tibialis for three weeks.	Recovery perfect.
6	10.2.28	6	4	40·5° C. (105° F.)	++++	+	Retention, photophobia, Amoss sign, pain in legs.	45	Lm.	++	750	50 I.V.	30 I.T. 30 I.V.	Right leg severely paralysed twelve hours later.	Delay of fifteen hours between lumbar puncture and injection of serum. The second dose of serum was given after onset of paralysis.
7	15.2.28	13	4, I.[1]	40° C. (104° F.)	+	+	Vomiting, pains in limbs.	180	60% Lm. 40% Pm.	+		15 I.T. 105 I.V.		No paralysis.	The intravenous injection was divided and given within twelve hours.
8	20.2.28	2	8, I.	38·3° C. (101° F.)	++	?	Photophobia, retention, drowsiness.	26	Lm.	+		10 I.T. 40 I.M.	10 I.M.	No paralysis.	General weakness for one month.
9	24.2.28 25.2.28	8	¾	37·4° C. (99·4° F.)	++ +++++	+ +	Photophobia, pains in legs, hyperæsthesia.	113 390	28% Lm. 72% Pm. 50% Lm. 50% Pm.	++ +++	755	30 I.V. 20 I.M.	20 I.T. 20 I.V. 30 I.M.	Paresis right gluteals for two months.	Recovery perfect.
10	27.2.28	3	3	38·3° C. (101° F.)	+++	+	Photophobia, hyperæsthesia, drowsiness.		"Increased Lm. and Pm."	+	760	15 I.T. 45 I.V.		Paralysis both legs.	A delay of thirty-six hours occurred between puncture and giving of serum.
11	4.3.28	12	1	39·4° C. (103° F.)	+++	+	Pain in back.	331	Lm.	++	710	18 I.T. 45 I.V. 5 I.M.		No paralysis.	Brother died of poliomyelitis four days before.
12	18.3.28	5	2	38·3° C. (101° F.)	+	+	Drowsy, irritable, photophobia, absent right knee jerk.	150	90% Lm.	+	740	19 I.T. 29 I.V. 12 I.M.	20 I.T. 20 I.V.	Paresis left quadriceps, right plantar flexors.	Recovery perfect.
13	19.3.28	7	3	38·7° C. (101·8° F.)	+	+	Drowsiness, pains in legs and neck.	250	92% Lm.	+		20 I.T. 20 I.M.	20 I.V.	No paralysis.	
14	19.3.28	7	3	37·8° C. (100° F.)	+++	+	Pains in legs, Amoss sign.	90	Lm., I.P.	+	740	30 I.T. 30 I.V.		Paresis left quadriceps for six weeks.	Recovery perfect.
15	2.4.28	7	2	38·3° C. (101° F.)	++	+	Vomiting, pains in legs, tremor of hands.		"Numerous M."	?		10 I.T. 10 I.M.		No paralysis.	Contact with 16 and 17.
16	9.4.28	6	1	38·3° C. (101° F.)	++	+	Malaise, tremor of hand.		"Numerous M."	?		10 I.T. 10 I.M.	10 I.T. 30 I.V.	Facial paresis on April 10, 1928.	Complete recovery in six weeks.
17	18.4.28	7	1	38·9° C. (102° F.)	+++	+	Tremor of hands, pain in back and neck.	150	"Almost all Lm."	?		10 I.T. 30 I.V.		No paralysis.	Contact with 15 and 16.
18	10.5.28	1	1	38·3° C. (101° F.)	++	?	Drowsy, irritable, vomiting, tremor of hands.	20	Lm.	++		9 I.T. 30 I.M.		No paralysis.	Contact with 15, 16, and 17.

[1] I. = Intermission occurred.
[2] Lm. = Lymphocytes; Pm. = Polymorphonuclear cells; M. = Mononuclear cells,
[3] B.S. = Blood-stained; I.P. = Increased pressure; N.L.P. = No lumbar puncture done.
[4] I.T. = Intrathecal; I.V. = Intravenous; I.M. = Intramuscular.

GROUP I.—PREPARALYTIC—*Continued.*

Group Number.	Date.	Age.	Duration of Illness in Days.	Temperature.	Spine Sign.	Headache.	Other Symptoms.	Cerebro-Spinal Fluid Findings.				Serum in c.cm.[4]		Result.	Comments.
								Number of Cells.	Type of Cells[2] or Description of Fluid.[3]	Globulin.	Chlorides in mgm. per 100 c.cm.	First Dose.	Second Dose.		
19	14.4.28	12	2	37·2° C. (90° F.)	++	+	Drowsiness, vomiting, pains in neck.	180	Lm	+	760	30 I.T. 30 I.V.	30 I.V. 30 I.M.	No paralysis.	
20	10.5.28	8	3	38·3° C. (101° F.)	+++	+	Sore throat, pains in leg and back.	100	(Approx.) Lm.	+	720	10 I.T. 5 I.M.		Death eight hours after serum.	Delay between puncture and serum.
21	18.5.28	11	¼	37·2° C. (99° F.)	+	+	Irritable, pains in limbs and back.	180	Lm., some Pm.	+	720	11 I.T. 14 I.M.		No paralysis.	
22	26.5.28	5	3	38·9° C. (102° F.)	++	+	Drowsy, tender hamstrings, Amoss sign.	120	95% Lm.	+	715	33 I.T. 47 I.V	30 I.M. (27.5.28) 30 I.M. (29.5.28)	Right facial, left hamstring, paresis for six weeks.	Recovery complete.
23	23.2.28	8	4	38·9° C. (102° F.)	+++	+	Vomiting, pain in neck, photophobia.	90	"Mainly Lm."	+	720	15 I.T. 45 I.V.	30 I.V.	Paresis left lower triceps for two months.	Recovery. Delay between puncture and serum.
24	11.6.28	37	4	38·5° C. (100·4° F.)	+++	+++	Photophobia, vomiting, drowsiness, pain in neck and back.	8	?	+		10 I.T. 20 I.V. 90 I.M.	50 I.M. (13.6.28) 40 I.M. (18.6.28)	Weakness right leg on seventh day.	Recovery complete.
25	22.6.28	3	5	38·9° C. (102° F.)	+++	++	Drowsiness, photophobia, opisthotonus.	20	Lm.	?	720	15 I.T. 40 I.M.		No paralysis.	Was backward before this illness.
26	11.28		1½	40·5° C. (105° F.)	++	+	Delirium, drowsiness, photophobia, vomiting.	200	60% Lm. 40% Pm.	+		18 I.T. 42 I.V.	40 I.V.	No paralysis.	Right basal pneumonia developed later.
27	12.1.29	2	5, I.[1]	37·8° C. (100° F.)	++	+	Irritable, falling over.	8[5]		?	760	26 I.T. 44 I.V.		Paresis right quadriceps, right peronei.	Recovery complete in two months.
28	23.1.29	4	5, I.	38·3° C. (101° F.)	++	+	Irritable, delirious sweating, Amoss sign.	5[5]		+	785	26 I.T. 64 I.V.		Paresis right quadriceps.	Recovery complete in three months. Brother paralysed.
29	28.1.29	4	2	38·4° C. (101·2° F.)	+++	+	Drowsy, vomiting, retention, pain in neck.	65	57% Lm. 43% Pm.	+	720	90 I.V.	30 I.T. 8 I.M.	Palate paresis for six days.	Recovery complete.
30	26.2.29	4	8, I.	38·3° C. (101° F.)	++	+	Photophobia, vomiting, apathy, pains in limbs.	276	Lm. and Pm.	+	710	17 I.T. 32 I.V.	20 I.T.	No paralysis.	
31	29.2.29	4	2	38·9° C. (102° F.)	++	+	Drowsy, vomiting.	180	65% Lm.	+	730	15 I.T. 25 I.M.	20 I.T. 10 I.V.	No paralysis.	
32	25.2.29	8	2½	38·9° C. (102° F.)	+++	+	Vomiting, retention, pains in back and abdomen.	40	Lm.	?	731	15 I.T. 25 I.V.	15 I.T. 25 I.M.	No paralysis.	Very toxic child.
33	9.3.29	7	2	38·3° C. (101° F.)	++	+	Drowsiness, pain in back.	70	Lm.	+		10 I.T.	40 I.V. (10.3.29) 10 I.T. (11.3.29) 10 I.V. (14.3.29)	Paresis right deltoid.	Almost complete recovery. Small initial dose.
34	16.3.29	10	1	38·5° C. (101·4° F.)	+++	+	Drowsy, Amoss sign.	40	"Mainly Lm."	+		9 I.T. 50 I.V.	30 I.V. (20.3.29)	Paresis right deltoid on fourth day.	Almost complete recovery.
35	16.3.29	5	10, I.	37·4° C. (99·4° F.)	+	+	"Influenza ten days before"; felt sick today.	27	Lm. and Pm.	+	730	10 I.T. 10 I.V. 10 I.M.		No paralysis.	Brother paralysed two weeks before. (Death.)
36	17.4.29	8	3	39·7° C. (103·6° F.)	+	+	Vomiting, drowsiness, abdominal pain.	120	Lm. and Pm.	+		10 I.T.	50 I.V. (18.4.29)	No paralysis.	

[1] I. = Intermission occurred.
[2] Lm. = Lymphocytes; Pm. = Polymorphonuclear cells; M. = Mononuclear cells.
[3] B.S. = Blood-stained; I.P. = Increased pressure; N.L.P. = No lumbar puncture done.
[4] I.T. = Intrathecal; I.V. = Intravenous; I.M. = Intramuscular.
[5] After standing.

GROUP I.—PREPARALYTIC—*Continued.*

Group Number.	Date.	Age.	Duration of Illness in Days.	Temperature.	Spine Sign.	Headache.	Other Symptoms.	Cerebro-Spinal Fluid Findings.				Serum in c.cm.[4]		Result.	Comments.
								Number of Cells.	Type of Cells[3] or Description of Fluid.[3]	Globulin.	Chlorides in mgm. per 100 c.cm.	First Dose.	Second Dose.		
37	25.2.29	9	1/2	38·9° C. (102° F.)	++	+	Drowsy, irritable, photophobia, pain in limb.	60	"Almost all Lm."	−		30 I.T.		No paralysis.	
38	13.3.29	1	2	38·9° C. (102° F.)	+++	?	Drowsiness, photophobia, vomiting, fever.	40	"Almost all Lm."	+		10 I.T. 30 I.M.		No paralysis.	
39	23.3.29	5	8, I.[1]	37·5° C. (99·6° F.)	++	+	Hyperæsthesia, irritable, pain in neck and spine.	36	"Mainly Lm."	+	700	12 I.T. 48 I.V.		No paralysis.	
40	23.3.29	5	7, I.	38·6° C. (101·6° F.)	+++	+	Drowsy, irritable, Amoss sign, hyperæsthesia.	150	Lm. and Pm.	++		20 I.T. 20 I.V.	30 I.V. (24.3.29) 30 I.V. (25.3.29)	Paresis of right face and right deltoid (24.3.29).	Recovery now complete. Small initial dose.
41	24.3.29	5	8	37·8° C. (100° F.)	+++	+	Constipation, epistaxis, pain in limbs, neck and back.	90	Lm.	?	760	30 I.V.		Paresis left quadriceps.	Delay of twenty hours between puncture and serum.
42	19.4.29	10	3	38° C. (100·4° F.)	++	+	Drowsiness, sweating.		N.L.P.	?		20 I.V.		No paralysis.	Three others of family paralysed during week. One died.

[1] I. = Intermission occurred.
[2] Lm. = Lymphocytes; Pm. = Polymorphonuclear cells; M. = Mononuclear cells.
[3] B.S. = Blood-stained; I.P. = Increased pressure; N.L.P. = No lumbar puncture done
[4] I.T. = Intrathecal; I.V. = Intravenous, I.M. = Intramuscular.

GROUP II.—EARLY PARALYTIC GROUP.

Group Number.	Date.	Age.	Duration of Illness in Days.	Temperature.	Spine Sign.	Headache.	Other Symptoms.	Cerebro-Spinal Fluid Findings.				Serum in c.cm.[3]		Result.	Comments.
								Number of Cells.	Type of Cells[1] or Description of Fluid.	Globulin.	Chlorides in mgm. per 100 c.cm.	First Dose.	Second Dose.		
1	24.7.27	2	1/2	37·5° C. (99·6° F.)	++	+	Drowsiness, weakness of right leg (thirty-six hours)	150	Lm.	—		20 I.T. 25 I.V. 15 I.N.		No spread of paralysis.	Has weakness of plantar flexors.
2	28.3.27	2 7/12	1 1/2	37·4° C. (99·4° F.)	+	?	Head falling forward, abdominal muscles.	20	Lm.	—		25 I.T. 15 I.M.		Weakness of abdominal muscles for three months.	No paralysis now.
3	12.11.27	7/12	1 1/2	38·9° C. (102° F.)	+	?	Right arm sore (one day), deltoid paralysed (twenty-four hours).	30	Lm.	—		70 I.M.		Weak deltoid for four months.	No paralysis now.
4	14.10.27	5/12	1	37·8° C. (100° F.)	++	?	Weakness of right arm (one day).	30	Lm.	—	720	40 I.M.		Weak deltoid for six months.	No paralysis now.
5	7.1.28	6	2	37·2° C. (99° F.)	+++	?	Paralysis of deltoid, arm muscles (twelve hours).	115	39% Lm. 61% Pm.	+		60 I.V.		Has severe deltoid weakness.	No further spread detected.
6	17.1.28	6	4	37·5° C. (99·6° F.)	+++	?	Palate paresis, drowsy, pain in neck.	110	Lm.	+		10 I.V. 40 I.M.		Abdominal muscles and right arm paresis for four months.	No paralysis now.

[1] Lm. = Lymphocytes; Pm. = Polymorphonuclear cells.
[2] I.T. = Intrathecal; I.V. = Intravenous; I.M. = Intramuscular.

GROUP II.—EARLY PARALYTIC GROUP—*Continued.*

Group Number.	Date.	Age.	Duration of Illness in Days.	Temperature.	Spine Sign.	Headache.	Other Symptoms.	Cerebro-Spinal Fluid Findings.				Serum in c.cm.[4]		Result.	Comments.
								Number of Cells.	Type of Cells[3] or Description of Fluid.[3]	Globulin.	Chlorides in mgm. per 100 c.cm.	First Dose.	Second Dose.		
7	10.2.28	15	10, I.[1]	37·8° C. (100° F.)	++	–	Photophobia, pain in right leg, weakness left hip muscles (one day).	50	Lm.	+	720	40 I.V. 30 I.M.		Had weak hip flexors for one year.	No paralysis now.
8	2.3.28	13	4	38·3° C. (101° F.)	++	–	Retention, weakness of neck muscles and of right arm (twelve hours).	48	94% Lm. 6% Pm.	+	750	25 I.T. 15 I.V.	20 I.V.	Spread to both legs two days later.	Sister paralysed four days before.
9	9.6.28	1	6/8	37·8° C. (100° F.)	+++	–	Drowsy, photophobia (two days), weak right arm (twelve hours).	210	95% Lm.	+	710	20 I.T. 20 I.M.	10 I.T. 35 I.M. (12.6.28)	Spread in right arm on June 12, 1928.	Delay of twelve hours after puncture. Residual right deltoid only.
10	10.7.28	4	4	39·4° C. (103° F.)	++	+	Photophobia, vomiting, pain in left leg, weakness of leg.	90	Lm.	+		15 I.T. 55 I.V.		Left quadriceps weak for six weeks.	No paralysis now.
11	26.7.28	7	4	37·9° C. (100·2° F.)	++	+	Vomiting, pain in neck, pain in limb, weakness of arm (one day).	150	Lm.	+		15 I.T. 35 I.V		Has weakness of left hand and abdominals only.	Arms and legs have recovered in three months.
12	4.1.29	4	2	38° C. (100·4° F.)	+	+	Unable to stand (one day), drowsy, irritable, paresis left quadricep.	25	Lm.	+		30 I.T. 30 I.V. 25 I.M.		No paralysis now.	Her brother paralysed same time.
13	8.1.29	4	1 1/8	38·5° C. (101·4° F.)	++	+	Stiff neck (thirty-six hours), right facial paralysis when serum was given.	300	Lm. and Pm.	++		25 I.T. 45 I.V.	20 I.M.	Paralysis of four limbs developed two days later. Recovery is widespread.	Delay of twelve hours between puncture and giving of serum.
14	26.1.29	10/12	3	37·2° C. (99° F.)	++	?	Paralysis of right quadricep, right buttock muscles (one day).			–		40 I.M.		No further spread.	Good recovery.
15	8.2.29	4	5	37·2° C. (99° F.)	++	?	Retention, photophobia, weakness of left foot muscle.	39	75% Lm. 25% Pm.	?		46 I.V. 18 I.T.		No extension.	Paresis of toes only now.
16	14.3.29	2	1 1/4	38·2° C. (100·8° F.)	++	?	Weakness of gluteal muscles and flexors of left foot		N.L.P.	–		50 I.V.		No further spread.	Slight residual weakness of ankle movement.
17	2.4.29	19	6		+++	?	Vomiting, pains in limbs, paralysis of right thigh (one day).	100	97% Lm. 3% Pm.	+		60 I.V.		No further spread. Residual paresis at three months, only one tibialis.	Will recover completely.
18	3.7.29	4	6	38·3° C. (101° F.)	++	+	Vomiting, constipation, left facial paresis (one day).	500	95% Pm. 5% Lm.			15 I.T. 25 I.M.		No further paralysis.	Face improving.
19	4.7.29	11	6	38·5° C. (101·4° F.)	+++	+	Vomiting, pains in limbs, opisthotonus, convulsions.	65	"Mostly Lm."	+	720	20 I.T. 20 I.V.		Transient paresis left face and arm.	Recovery complete.

[1] I. = Intermission occurred.
[2] Lm. = Lymphocytes; Pm. = Polymorphonuclear cells.
[3] N.L.P. = No lumbar puncture done.
[4] I.T. = Intrathecal; I.V. = Intravenous; I.M. = Intramuscular.

SYNOPSIS OF CASES TREATED BY HUMAN IMMUNE SERUM.

GROUP IV.—SPREADING TYPE.[5]

Group Number	Date.	Age.	Duration of Illness in Days.	Temperature	Duration of Paralysis in Days.	Extent of Paralysis.	Symptoms.	Spine Sign.	Cerebro-Spinal Fluid Findings.			Serum in c.cm.[4]		Result.	Comment.
									Number of Cells.	Type of Cells[2] or Description of Fluid.[3]	Globulin.	First Dose.	Repeat Dose.		
1	2.1.28	6	7	37·2° C. (99° F.)	1 (right leg). 4 (palate)	Right leg severely.	Vomiting, pain in neck and leg.	+	250	66% Lm. 44% Pm.	+	50 I.V.		No spread.	End result good. Will walk well.
2	*22.1.28*	*6*	*2*	*38·9° C. (102° F.)*	*1 1/2*	*Both legs, both arms.*	*Vomiting, headache.*	++		*N.L.P.*	–	*20 I.T. 20 I.V.*		*Death from respiratory paralysis on January 24, 1928.*	*Cousin and contact of 3.*
3	*28.1.28*	*7*	*4*	*37·2° C. (99° F.)*	*1 1/2*	*Both arms, right leg.*	*Headache, fever, pain in back.*	++	*120*	*60% Lm.*	?	*30 I.V.*		*Died two days later. She had respiratory involvement on admission.*	*Contact of 2.*
4	26.1.28	19	6	39 1° C. (102·4° F.)	3	All four limbs.	Headache, drowsiness, retention.	+++	180	Lm.	–	50 I.V.		No further spread.	
5	*30.1.28*	*14*	*4*	*38° C. (100·4° F.)*	*2*	*Paralysis of both arms and intercostal muscles.*	*Headache, drowsiness, vomiting.*	+++	*70*	*96% Lm.*	+	*50 I.V.*		*Death from respiratory paralysis (thirty hours later).*	
6	18.2.28	9	4	38·3° C. (101° F.)	2	All four limbs severely.	Vomiting, headache.	++	110	89% Lm.	–	20 I.T. 10 I.V.	10 I.T.	No spread of paralysis noticed after serum.	
7	*27.2.28*	*56*	*14*	*38·3° C. (101° F.)*	*1*	*Right leg.*	*Fever, headache.*	+++		*B.S.*	–	*20 I.T. 25 I.V.*	*15 I.M.*	*Death two days later after spread to other leg.*	
8	*27.6.28*	*8*	*3*	*39·7° C. (103·6° F.)*	*1*	*Both arms, neck muscles.*	*Vomiting.*	+		*N.R.*		*50 I.V. 25 I.T.*	*30 I.V.*	*Death from respiratory paralysis.*	*The injections were followed by hypertonic saline solution given intravenously.*
9	2.3.28	14	17	38·7° C. (101·8° F.)	7	Left leg severely.	Pain in right deltoid on the ninth after seven days' fever.	+	80	B.S.	?	20 I.T., 20 I.V., given on the 9th.	20 I.V. on 20.3.28	No further paralysis.	"Jump" type of Batten.
10	24.4.28	4	4	38° C. (100·4° F.)	2 1 1/2	Right leg. Left leg. Right arm.	Headache, vomiting, pains in legs and back.	++	80	Lm.	+	20 I.T. 40 I.V.	45 I.V.	Abdominal muscles were involved between the two injections.	Sister also paralysed.
11	*14.6.28*	*3*	*3*	*38·6° C. (101·6° F.)*	*1 1/4*	*Left arm, left leg.*	*Pain in the neck, fever, drowsiness.*	+		*B.S.*	–	*10 I.T.*		*Death seven hours later.*	
12	3.1.29	6	4	38·7° C. (101° F.)	1 1/2 1/2	Left arm. Right leg.	Headache, vomiting.	++	200	95% Lm. 5% Pm. I.P.	+	25 I.T. 75 I.V.		No further spread.	
13	9.2.29	30	10	38·9° C. (102° F.)	4 1/2	Left leg. Left arm.	Vomiting, pains in back and knees.	+++		N.L.P.	–	50 I.V.		No further spread.	Good recovery occurring.
14	*4.4.29*	*8*	*3*	*38·9° C. (102° F.)*	*1*	*Right face, both arms, palate.*	*Abdominal pain, inability to swallow.*	++		*N.L.P.*	–	*70 I.V.*		*Death three days later. Respiratory.*	
15	19.4.29	9	5	38·3° C. (101° F.)	1 1/2	Right arm, right leg.	Headache, vomiting, retention.	++	110	Lm.	+	30 I.T.		Left arm, left leg affected six hours later.	
16	*7.4.29*	*16*	*8*	*38·3° C (101° F.)*	*2* *1 1/2* *1/2*	*Both legs.* *Both arms.* *Abdomen, face.*	*Headache, vomiting, retention, pain in back.*	++	*180*	*Lm.*	+	*15 I.T. 55 I.V.*	*20 I.V. 10 I.M.*	*Death four days later.*	*Spread after serum.*

[1] I. = Intermission occurred.
[2] L.m. = Lymphocytes; Pm. = Polymorphonuclear cells.
[3] N. L. P. = No lumbar puncture done; B.S. = Blood-stained; N.R. = No record; I.P. = Increased pressure.
[4] I.V. = Intravenous; I.T. = Intrathecal; I.M. = Intramuscular.
[5] The particulars in regard to patients who died, are printed in italics.

GROUP IV.—SPREADING TYPE[1]—*Continued.*

Group Number.	Date.	Age.	Duration of Illness in Days.	Temperature.	Duration of Paralysis in Days.	Extent of Paralysis.	Symptoms.	Spine Sign.	Cerebro-Spinal Fluid Findings.		Serum in c.cm.[4]		Result.	Comment.
									Number of Cells.	Type of Cells[5] or Description of Fluid.[6]	Globulin.	First Dose.	Repeat Dose.	
17	30.3.29	18	8	39·4°C. (103°F.)	2 2 1	Two legs. Arms. Abdominal muscles and diaphragm.	Headache, pain in back and abdomen.	++		N.L.P.	–	50 I.V.	50 I.V. (1.4.29)	No further spread after second dose of serum.
18	4.5.29	18	5	37·5°C. (99·6°F.)	2 1/2	Both legs. Left arm.	Vomiting, headache, pains in back and abdomen.	++	250	Lm.	+	18 I.T. 62 I.V.	30 I.M. 30 I.M.	No further spread, arm clearing up quickly.	Desquamation. Activity in early stage.
19	29.2.28	17	7	38·9°C. (102°F.)	3 1	Both legs. Right arm.	Fever, retention.	++		N.L.P.	–	50 I.V.		No further spread.	Arm cleared up quickly.
20	12.11.25	10	14, I.[1]	37·8°C. (100°F.)	5 4 1	Right leg. Left leg. Right arm.	Headache, drowsiness, vomiting, retention.	++		B.S.	–	15 I.T. 15 I.V.		No further spread.	
21	13.3.26	13	4	38·3°C. (101°F.)	3 1/2	Both arms. Right leg.	Pains in limbs, retention.	++	90	Lm.	+	12 I.V. 33 I.M.		No further spread.	
22	26.10.26	15	7		4 2 1	Both legs. Arms. Swallowing, breathing.	Vomiting, headache, backache.	++		B.S.	–	10 I.T. 40 I.V. 20 I.M.	50 I.M.	No further spread.	Complete recovery in nine months.

[1] I. = Intermission occurred.
[2] Lm. = Lymphocytes; Pm. = Polymorphonuclear cells.
[3] N.L.P. = No lumbar puncture done; B.S. = Blood-stained; N.R. = No record; I.P. = Increased pressure.
[4] I.V. = Intravenous; I.T. = Intrathecal; I.M. = Intramuscular.
[5] The particulars in regard to patients who died are printed italics.

Group II.

Dr. Allan, Case 6.
Dr. Armstrong, Case 11.
Dr. Brauer, Case 18.
Dr. Campbell, Cases 3, 14.
Dr. Johnston, Case 8.
Dr. McMahon, Case 5.
Dr. Newman, Case 7.
Dr. A. H. Norris, Cases 9, 12, 13, 16.
Dr. Patrick, Case 18.
Dr. Sheahan, Case 10.
Dr. Spencer, Case 19.
Dr. Wilson, Case 19.
Children's Hospital (Dr. Ferguson, Dr. Forsyth, Dr. Grieve), Cases 1, 2, 4, 5.
Melbourne Hospital (Dr. Cooper), Case 17.

Group IV.

Dr. Burwood, Case 16.
Dr. Connor, Case 19.
Dr. Davies, Case 12.
Dr. Fetherston, Case 15.
Dr. Fitzpatrick, Case 19.
Dr. Grieve, Cases 12, 21.
Dr. Langmore, Case 9.
Dr. Pryde, Case 22.
Dr. Rutter, Case 17.
Dr. Scott, Case 18.
Dr. Skelton, Case 11.
Dr. Sutcliffe, Case 7.
Children's Hospital (Dr. Ferguson, Dr. Forsyth), Cases 1, 2, 3, 6, 8, 20.
Melbourne Hospital (Dr. Cowan, Dr. Hiller), Cases 4, 5, 10.
Alfred Hospital (Dr. Major, Dr. Norris), Cases 13, 14.

References.

(1) S. Flexner and P. A. Lewis: "Experimental Poliomyelitis in Monkeys," *Journal of the American Medical Association*, May 28, 1910, page 1781.

(2) Levaditi and Landsteiner: *Comptes Rendus des Séances de la Société de Biologie*, Volume LVIII, 1910, page 311.

(3) A. Netter: *Bulletin de l'Académie de Médecine*, January 31, 1928, page 147.

(4) H. L. Amoss and A. M. Chesney: "A Report on the Serum Treatment of Twenty-six Cases of Epidemic Poliomyelitis," *Journal of Experimental Medicine*, Volume XXV, 1917, page 581.

(5) George Draper: "Acute Poliomyelitis," 1917.

(6) W. S. Robertson: "A Review of the Cases of Polioencephalitis in the Wellington Hospital," *New Zealand Medical Journal*, October, 1925, page 235.

(7) C. M. Hector: "Poliomyelitis," "Transactions of the Australasian Medical Congress (British Medical Association)," Second Session, Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1927, page 258.

(8) F. G. Morgan: "Collecting Blood from Donors and Preparation of Curative Serum for Use in Poliomyelitis," "Transactions of the Australasian Medical Congress (British Medical Association)," Second Session, Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1927, page 263.

(9) Jean Macnamara: "Treatment of Poliomyelitis by Means of Human Immune Serum," "Transactions of the Australasian Medical Congress (British Medical Association)," Second Session, Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1927, page 265.

(10) W. D. Ayer: "Poliomyelitis: Its Preparalytic Period, with Results of Serum Therapy," *American Journal of the Medical Sciences*, Volume CLXXVII, Number 4, April, 1929, page 540.

(11) W. L. Aycock and E. H. Luther: "Preparalytic Poliomyelitis," *The Journal of the American Medical Association*, August 11, 1928, page 387.

(12) W. L. Aycock and H. L. Amoss: "Experiments on Local Specific Therapy in Poliomyelitis: The Utilization of Hypertonic Solutions in the Serum Treatment of Experimental Poliomyelitis," *Bulletin of the Johns Hopkins Hospital*, Volume XXXIV, 1923, page 361.

(13) Reginald Webster: "Lumbar Puncture and Meningitis," THE MEDICAL JOURNAL OF AUSTRALIA, November 27, 1920, page 487.

(14) S. Flexner and H. L. Amoss: *Journal of Experimental Medicine*, Volume XXV, 1917, page 525.

(15) George M. Lyon: "Cytolysis in the Cerebro-Spinal Fluid in Acute Poliomyelitis," *American Journal of Diseases of Children*, July, 1928, Volume XXXVI, Number 1.

(16) Shaw, Thelander and Fleischner: "Convalescent Serum in Preparalytic Cases of Poliomyelitis," *The Journal of the American Medical Association*, November 14, 1925, page 1555.

THE ACTIVITY OF STORED ANTIPOLIOMYELITIC SERUM IN EXPERIMENTAL POLIOMYELITIS.[1]

By F. M. BURNET,[2] M.D. (Melbourne), Ph.D. (London),

AND

JEAN MACNAMARA, M.D. (Melbourne),

From the Walter and Eliza Hall Institute for Research in Pathology and Medicine, Melbourne Hospital.

THE fresh serum from convalescent human patients with poliomyelitis has been conclusively shown to possess preventive and therapeutic activity in experimental poliomyelitis in the monkey, first by Flexner and Lewis[(1)] in 1910, afterwards by many others. The use of such serum in preparalytic and early paralytic cases in human beings has therefore a rational basis and clinically has given excellent results.

If this method of treatment is to be made generally available, it is obvious that some method of preparing the convalescent serum must be developed which will allow of its being available at the shortest notice. Particularly in Australia with its large distances and scattered population, dependence on the presence of suitable convalescent patients in the locality is impossible in country districts where a majority of the infections has occurred, and very impracticable in the cities. Stocks of sterile serum in suitable containers must be kept available at suitable centres in city and country.

To allow this the technique was adopted by Dr. F. G. Morgan[(2)] and his coworkers at the Commonwealth Serum Laboratories of preparing the serum from oxalated plasma according to the methods used with commercial horse antisera. Sterility was assured by the use of a rigidly aseptic technique and filtration through porcelain candles. The product was finally sealed in ampoules without added antiseptic and kept stored at ice box temperature.

This technique is more elaborate than any reported from America or France and it seemed of the first importance that direct experimental proof should be available that the various manipulations had not destroyed the virucidal power of the fresh serum. In most forms of serum therapy the administration of inactive serum is not likely to do positive harm, but in an early case of poliomyelitis it is essential that nothing but active immune serum be introduced into the spinal theca. Flexner and Amoss[(3)] (1917) have shown that in monkeys that have received poliomyelitis virus intravenously, intranasally or subcutaneously, the introduction of aseptic normal serum or saline solution will promote infection of the central nervous system. The condition of these monkeys is closely analogous to that of a child in the preparalytic stage of poliomyelitis and in some cases irretrievable harm might be done by the clinical administration of an inactive serum. If the clinical work is to have a sound experimental basis, it is necessary to determine first that the serum as prepared is actively virucidal and secondly how long the product can be kept in cool storage and still retain most of its activity.

In the experimental work attention was confined to the so-called virucidal properties of the serum, that is, its power to render an infective filtrate or suspension innocuous when injected intracerebrally. The difficulty of handling monkeys as well as the large amounts of serum needed made it impracticable to attempt any therapeutic experiments on monkeys in the late incubation period. This theoretically is the only type of experiment that can give any real analogy to the conditions in which the serum is used in practice. But from the work of Flexner and Lewis in 1910 it can be taken that sera show the same relative efficiency in the two types of experiment and for the purpose in hand the former technique was considered adequate.

The material for the first series of experiments was derived from a patient dying at the Children's Hospital (E.B.).

Clinical Notes.

E.B., a boy, aged five years and nine months, was admitted to the Children's Hospital on February 21, 1928, complaining of inability to stand. He had been well until two days previously, when he complained of a frontal headache. Next day he vomited several times and remained in bed, but no weakness of the legs was noticed until the day of admission.

On examination the patient looked flushed and ill with a dirty tongue and reddened throat. General examination of chest and abdomen revealed no abnormality. There was no definite head rigidity, but the patient was more comfortable when the head was allowed to fall back. The hips could not be flexed and there was great loss of power to extend the knees, although the *quadriceps* muscles were not completely inactive. Neither knee perk was elicited. He could extend both ankles and the ankle jerks were equal and active. The right foot could be dorsiflexed, but not the left.

On February 22, 1928, the condition was worse, with extension of the paralysis to most of the muscles of the right arm and shoulder and to the left triceps. The cerebro-spinal fluid obtained was too blood stained for examination. Fifty cubic centimetres of antipoliomyelitic serum were administered intrathecally and intravenously.

On the following day there was a further extension involving the muscles of respiration with paralysis of the intercostals and early on the morning of February 24, 1928, death occurred from asphyxia.

This was therefore a typical case of the acute ascending spinal type of anterior poliomyelitis.

The cord was removed as aseptically as possible and stored in 50% glycerin. An emulsion in saline solution, roughly 5%, was injected intracerebrally into a *Macacus rhesus* (Monkey 1). The animal showed typical symptoms and was killed on the ninth day. The spinal cord was removed aseptically and stored in sterile 50% glycerin in the ice chest.

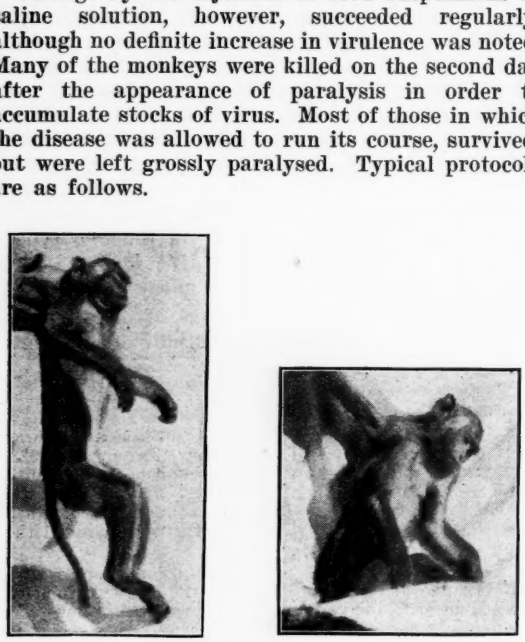
Four months later a 5% emulsion in saline solution of this cord (Monkey 1) was centrifuged and the supernatant fluid passed through a Seitz filter.

[1] Read at the Australasian Medical Congress (British Medical Association), Third Session, Sydney, September, 1929.

[2] Working under a grant from the Commonwealth Department of Health.

One cubic centimetre of the filtrate was injected intracerebrally into Monkey 4. At the same time a similar amount of unfiltered suspension was injected intracerebrally into Monkey 3. Both animals showed typical poliomyelitis. Monkey 3 showed paralysis on the ninth day, Monkey 4 on the twelfth. The cords of both were removed to glycerin. Several attempts were made to continue passage by means of filtrates, but these were unsuccessful.

Passage by the injection of cord suspensions in saline solution, however, succeeded regularly, although no definite increase in virulence was noted. Many of the monkeys were killed on the second day after the appearance of paralysis in order to accumulate stocks of virus. Most of those in which the disease was allowed to run its course, survived, but were left grossly paralysed. Typical protocols are as follows.



FIGURES I AND II.
Monkeys showing almost complete flaccid paralysis six days after intracerebral inoculation of Rockefeller virus.

MONKEY 6.—On January 11, 1929, 0·5 cubic centimetre of suspension of the cord of Monkey 3 was injected intracerebrally. On January 21, 1929 (the tenth day), the animal had weakness in both legs and was unable to stand without support. There was little change on the next day, but on January 23, 1929, both lower limbs were completely paralysed. The monkey could sit up in its cage and the arms appeared to be normal. On January 28, 1929, the left leg could be used slightly.

MONKEY 7.—On January 11, 1929, 0·5 cubic centimetre of suspension of the cord of Monkey 4 was injected intracerebrally on the right side. On January 14, 1929, a spastic paralysis of the left arm was noticed with a slighter degree of weakness in the left leg and left facial muscles. This was almost certainly due to trauma inflicted by the injection. On the tenth day the left leg seemed decidedly worse and its paralysis more flaccid than spastic. Next day both legs were completely paralysed and the left arm was weaker and flaccid. On the thirteenth day the monkey lost its power to sit up in the cage, but the right arm was still unaffected. The animal appeared otherwise healthy and ate well. There was slight recovery of power in the right leg during the next week, but otherwise the condition remained stationary.

MONKEY 14.—The animal was injected intracerebrally with a mixture of pooled virus, 0·5 cubic centimetre, and normal human serum, 0·5 cubic centimetre, on January 29, 1929. On the eleventh day the monkey was excitable with a tremor in the head movements and in the right leg, particularly when standing. The fur was ruffled over the back and shoulders. The next day both legs were partially paralysed and on the thirteenth day paralysis of the legs and the left arm was almost complete. The animal could not sit up and lay helpless on its side. The right arm was affected next day and thereafter the condition remained unaltered till the monkey died on the twentieth day.

Protection Experiments.

In the absence of a reliable filtrate the virus used in the protection experiments was prepared by grinding up two small pieces of preserved cord from each of four monkeys (4, 8, 12, 13) with saline solution to give an approximate 5% to 10% suspension. By this means it was hoped to have an approximately standard virus for all the experiments contemplated. As will be seen from the protocols, this expectation was not completely fulfilled. However, virus prepared in this way invariably produced unmistakable poliomyelitis if it was injected without immune serum. The results obtained in all monkeys injected with 0·5 cubic centimetre of virus without immune serum are shown in Table I. Owing to shortage of monkeys, animals that had been protected in one experiment were occasionally used as controls in a subsequent one. In accordance with the findings of all workers on the subject such animals showed no trace of immunity. Animals twice injected in this way are marked with an asterisk.

TABLE I.

Monkey	Day of First Paralysis.	Outcome.
3	9	
*6	10	
*7	12	
8	13	
*9	7	
*10	7	
12	10	
13	10	
14	10	Died on twentieth day.
*15	—	Died of tuberculosis on tenth day. No paralysis noted.
*16	9	Survived.
*21	8	Died on ninth day.
29	9	
30	9	

Although the virus is of only moderate activity, it will be seen that it is capable of causing typical paralysis in at least 90% of monkeys. Failure to develope poliomyelitis after injection of mixtures of virus and immune serum can therefore be taken as valid evidence of the presence of virucidal antibodies in such serum. In all protection experiments a fresh emulsion of pooled cords was made in saline solution to about 5% strength. The larger particles were avoided by taking up the emulsion in a very fine pointed capillary pipette. Mixtures with immune or normal serum were made as required and left overnight in the ice chest before being injected intracerebrally.

All experiments were done under full ether anæsthesia and aseptically. The monkey's head was shaved and the skin cleaned with lysol and spirit.

The area was surrounded with sterilized towels and a semicircular incision made in the skin over the right frontal region. The periosteum and overlying fascia were cleared from a sufficient area and a circle of bone about a centimetre in diameter was removed with a small trephine, care being taken not to damage the *dura mater*. The needle was introduced through the *dura* away from any large veins and the injection was made into the substance of the brain at a depth of about 1·5 centimetres. Even with very slow injection it was found impossible to prevent a certain amount of leaking along the needle track when the needle was withdrawn, but this loss was fairly uniform in the different monkeys and can probably be disregarded. The periosteum and fascia were now drawn over the opening and held by one or two sutures and the skin incision was closed with sutures of linen thread. The general health of the animals was usually quite unaffected by these procedures. There were no cases of meningitis or scalp infection. In one a moderately large hæmatoma formed and in two there was a partial hemiplegia for the first few days after inoculation. When the animals were examined *post mortem* at the height of their paralysis, the site of injection occasionally manifested no abnormality, but more usually there was a small cyst with a ragged wall and containing brownish fluid.

Protocols of the protection experiments may now be given.

EXPERIMENT I (January 29, 1929).—The virus used was a suspension from the pooled cords of Monkeys 8, 9, 10. Normal serum was obtained from the umbilical cord blood of three infants as being unlikely to show any antivirus properties. The serum was pooled and filtered through a Seitz filter. The immune serum was recently prepared (Batch 26, Group I, January, 1929). The disposition of the experiment and its results are shown in Table II.

TABLE II.

Protection by Immune Serum.

Monkey.	Virus in c.cm.	Normal Serum in c.cm.	Immune Serum.	Outcome.
14	0·5	0·5	—	Paralysed tenth day. Died twentieth day.
15	0·25	0·75	—	No paralysis.
16	0·5	—	0·5	No paralysis.
17	0·5	—	0·5	No paralysis.
18	0·25	—	0·75	No paralysis.
19	0·25	0·5	0·25	No paralysis.

This experiment shows that the immune serum can neutralize 0·5 cubic centimetre of virus which is quite active, in the presence of normal serum. Half the amount of virus, however, when mixed with three times its volume of normal serum, failed to cause paralysis, so that the apparent protection of Monkeys 18 and 19 is of no significance.

EXPERIMENT II.—Pooled virus from cords 4, 8, 12, 13 was used. Immune sera used were Group I, Batch 26 and Group II, prepared in April, 1928, and stored at ice chest temperature in the country. The results are shown in Table III.

TABLE III.

Monkey.	Virus in c.cm.	Serum Group I in c.cm.	Serum Group II in c.cm.	Saline Solution in c.cm.	Outcome.
15	0·5	—	—	0·5	Died of tuberculosis on the tenth day. No paralysis was noted.
16	0·5	—	—	0·5	Paralysed ninth day. Survived.
20	0·5	0·5	—	—	Died ninth day. Cellulitis of abdominal wall, etc.
21	0·5	0·25	—	0·25	No paralysis.
22	0·5	0·1	—	0·4	No paralysis.
23	0·5	—	0·5	—	No paralysis.
24	0·5	—	0·25	0·25	No paralysis.
25	0·5	—	0·1	0·4	No paralysis.

From this experiment it seems that 0·1 cubic centimetre of either serum will protect against 0·5 cubic centimetre of virus.

EXPERIMENT III.—In this the survivors from Experiment II were used eighteen days after the previous injections. Virus and immune sera were as before.

TABLE IV.

Monkey.	Virus in c.cm.	Serum Group I in c.cm.	Serum Group II in c.cm.	Outcome.
21	0·5	—	—	Paralysed eighth day. Died ninth day.
22	0·5	0·1	—	No paralysis.
23	0·5	—	—	Paralysed eighth day.
24	0·5	—	—	Paralysed tenth day.
25	0·5	—	0·1	No paralysis. Died on thirteenth day with great wasting, cause unknown.

It is unfortunate that in this experiment Monkey 25 died from other causes, while there was still a possibility of its developing poliomyelitis, but the regularity of paralysis on the eighth to the tenth day amongst the controls makes it likely that in both Monkeys 22 and 25 0·1 cubic centimetre of serum was sufficient to render the virus innocuous.

EXPERIMENT IV.—In the final experiment with local virus an attempt was made to determine an endpoint to the protective power of Group I serum. Virus and serum were as before.

TABLE V.

Monkey.	Virus in c.cm.	Serum Group I in c.cm.	Outcome.
26	0·5	0·25	Paralysed twenty-third day, paralysis almost confined to hip muscles.
27	0·5	0·1	Paralysed tenth day, severe.
28	0·5	0·05	Died half hour after recovery from anæsthetic.
29	0·5	—	Paralysed ninth day, severe.
30	0·5	—	Paralysed ninth day, complete prostration.
31	0·5	0·025	Paralysed tenth day, moderately severe.

This experiment illustrates the difficulty of attempting to titrate the activity of an antipoliomyelitic serum when the infecting dose of virus cannot be standardized. The paralyses in this batch were rather more severe than usual and it is presumed that the pieces of cord chosen happened to contain more virus than the average. It is clear that Monkey 26, with slight paralysis twenty-three days after inoculation, was almost completely protected by 0·25 cubic centimetre of serum, but the smaller amounts were quite without effect, although

in two previous experiments 0·1 cubic centimetre had protected against a similar dose of virus.

A similar experiment was planned for Group II serum, but owing to shortage of animals only two serum dilutions were used, 0·1 and 0·05 cubic centimetre. Two previously injected monkeys, 22 and 17, received these amounts along with 0·5 cubic centimetre of the virus suspension used in Experiment IV. The first showed no paralysis, the second was paralysed on the ninth day.

We may summarize the protection experiments in a single table showing the fate of monkeys inoculated with 0·5 cubic centimetre of suspension virus and different amounts of immune sera. In the table P. denotes paralysis, the figure following it giving the day on which paralysis was first noted. N. signifies no paralysis, D. died from unrelated causes on the day shown.

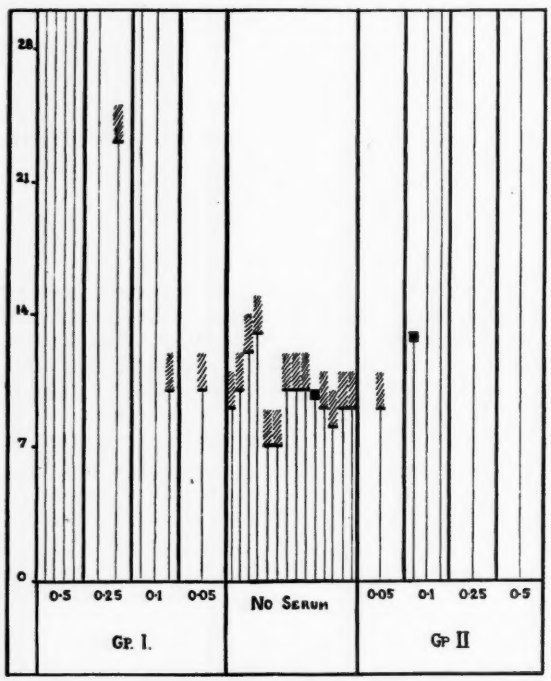


FIGURE III.

Graphic representation of protection experiments in which local virus was used. Each fine line represents a monkey inoculated with 0·5 cubic centimetre of suspension and the amount of serum shown. The hatched rectangles denote the period in days at which paralysis developed, a black square signifies death from some intercurrent disease.

Protection Experiments with Rockefeller Virus.

In June, 1929, we received from the Rockefeller Institute the cord of Monkey 2,187, dated February 5, 1929, and later a letter from Dr. Flexner explaining that the strain was highly virulent, being effective in the dosage of 0·005 cubic centimetre of filtrate before transport to Australia.

During June and July this virus was passed through monkeys until we found that 0·5 cubic centimetre of filtrate produced paralysis on the fifth to the sixth day.

The filtrate prepared from the cord of Monkey 35 has been used in the protection experiments, protocols of which may now be given.

TABLE VI.

Monkey.	Rockefeller Virus (Filtrate M35).	Serum.	Group and Date.	Outcome.
36	0·5	—	—	Paralysis on sixth day. Died seventh day.
37	0·5	—	—	Paralysis on sixth day. Killed seventh day.
38	0·5	0·1	Group I, 1929	No paralysis.
39	0·5	0·2	Group I, 1929	No paralysis.
40	0·5	0·5	Group I, 1929	No paralysis.
41	0·5	0·5	Group I, 1929	No paralysis.

This serum was recently prepared serum from recent donors.

TABLE VII.

Monkey.	Rockefeller Virus (Filtrate M35).	Serum.	Group and Date.	Outcome.
42	0·5	—	—	Paralysis on fifth day. Died sixth day.
43	0·05	—	—	Died intercurrent fourth day.
44	0·5	0·5	Group I, Jan. 1926	No paralysis.
45	0·5	0·25	Group I, Jan. 1926	No paralysis.
46	0·5	0·5	Group II, Jan. 1928	No paralysis.
47	0·5	0·25	Group II, Jan. 1928	No paralysis.
48	0·5	0·1	Group I, Jan. 1929	No paralysis.

The last experiment has been to compare the activity of serum prepared from 1925 donors, taken soon after the illness, in January, 1926, with that from donors in the same group taken in April, 1929.

TABLE VIII.

Showing Result in Table VII.

Monkey.	Rockefeller Virus (Filtrate M35).	Serum.	Group and Date.	Outcome.
49	0·5	—	—	Paralysis fifth day. Killed sixth day.
50	0·5	0·2	Group I, Jan. 1926	No paralysis.
51	0·5	0·1	Group I, Jan. 1926	Paresis left arm.
52	0·5	0·2	Group II, April 1929	No paralysis.
53	0·5	0·1	Group II, April 1929	No paralysis.
40	0·5	—	—	Paralysis fifth day. Died sixth day.

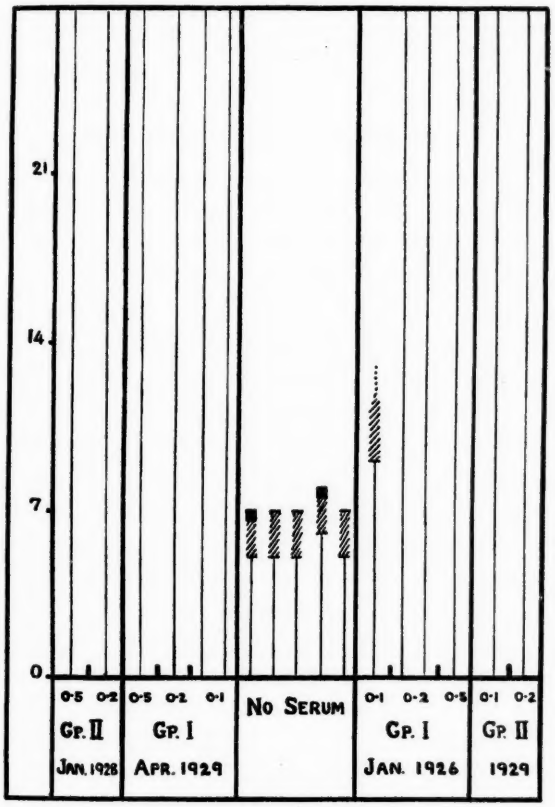
It appears that the serum prepared in January, 1926, and stored in ampoules has slightly weaker protective power than that prepared from the same persons in 1929.

The experiments with this stronger virus are summarized in Figure IV.

Discussion.

The aim of the present experiments was first to make certain that the filtered and stored convalescent serum as used therapeutically still retained virucidal antibodies and secondly to compare the activity of sera of Groups I and II, that is sera

from persons recently convalescent (up to one year), with sera from persons infected at a more distant period. The experiments reported are sufficient to show that the serum, as prepared and distributed by the Commonwealth Serum Laboratories, is actively virucidal and protects monkeys against intracerebral inoculation of both a locally obtained virus and a highly active one of American origin. The attempt to obtain a definite value for the minimum protective dose of the two types of sera must be considered to have failed. As far as the two batches examined are concerned, the serum from the older cases (Group



ROCKEFELLER VIRUS.

0·5 cc FILTRATE.

FIGURE IV.

To summarize protection experiments made with Rockefeller virus. Signs as in Figure III, but in this table the outcome of paralysis is represented, natural death by a black square, killed when moribund by a horizontal line and survival by a dotted continuation of the line.

II) seems slightly more potent than that from more recently recovered patients, but the number of experiments is far too small to allow us to substantiate this. Like other observers we have found that practically all monkeys are susceptible to the virus, so that individual variation of animals is probably of less significance in this work than in most *in vivo* titrations of antisera. The most important uncontrolled factor is the activity and concentration of the virus. As fresh suspensions must be made up from the cords stored in glycerin for each experiment, it is impossible to be sure that the dosage used in different experiments is comparable. The difference between the results in Experiments II and IV is clearly attributable to differences in the dosage of virus, although fragments from the same cords were used in both experiments.

The use of the American virus in the form of a filtrate has enabled us to avoid this difficulty. The results of the second series of experiments are quite consistent among themselves, all the sera being active in doses of 0·1 cubic centimetre, except one very old sample which gave only partial protection in this amount. It must be recognized that virus in the form of a filtrate will probably be much more readily inactivated than the same amount in a thick suspension of cord substance. This is almost certainly sufficient to account for the apparent greater sensitivity of the Rockefeller virus to the neutralizing action of the serum.

Conclusions.

1. Human immune serum prepared by the technique adopted by the Commonwealth Serum Laboratories for the organization in Melbourne is effective in protecting monkeys against intracerebral inoculation of active poliomyelitic virus. This technique involves treatment with oxalate and filtration. No tricresol or other antiseptic is added.

2. The virucidal qualities are retained for at least three years when the ampouled serum is stored at ice box temperature.

3. No significant difference was detected between the virucidal properties of sera prepared from (i) persons recently convalescent and (ii) persons affected three years previously.

4. A method of therapy is therefore available which rests upon a sound experimental basis.

Acknowledgements.

The use of monkeys for this experimental work was rendered possible by a grant from the Commonwealth Government, Department of Health.

In addition we have to thank Dr. C. H. Kellaway for assistance and criticism, and Mr. Wilkie, Director of the Zoological Gardens, for his help in obtaining monkeys.

For the Rockefeller virus we are greatly indebted to Dr. Simon Flexner, and finally we have to thank Dr. George Simpson for obtaining cinema films of representative patients, human and simian.

References.

(1) S. Flexner and P. A. Lewis: "Experimental Poliomyelitis in Monkeys," *Journal of the American Medical Association*, May 28, 1910, page 1781.

(2) F. G. Morgan: "Collecting Blood from Donors and Preparation of Curative Serum for Use in Poliomyelitis," "Transactions of the Australasian Medical Congress (British Medical Association)," Second Session, Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1927, page 263.

(3) S. Flexner and H. L. Amoss: *Journal of Experimental Medicine*, 1917, Volume XXV, page 525.

Reviews.

TREATMENT BY PHYSICAL MEANS.

IN his "Physical Therapeutic Technic" Dr. Frank B. Granger writes especially for the general practitioner who does a certain amount of this work.[1] There is a detailed survey of all the various physio-therapeutic measures, outlining the principles of each and the method of their application. This part is well set out and lucidly written. The latter half of the book is devoted to treatment of the individual diseases. This is very well arranged for rapid reference, but the clinical conditions dealt with are too encyclopædic. In many cases the claims set out for physiotherapy are excessive. The inclusion of such conditions as amenorrhœa, diabetes, coryza, eye strain, biliary and renal colic, impotence and many others, detracts from an otherwise useful book. The illustrations are good, but are arranged without any reference to the letterpress.

SURFACE ANATOMY.

THE seventh edition of "Landmarks and Surface Markings of the Human Body," by L. Bathe Rawling, is a small, handy volume of eighty-five pages of actual reading matter which doubtless fills a gap in a somewhat neglected branch of our medical education.[2] The author's main aim is simplification, so much so that only such landmarks are given as are of practical value and each landmark is independent of any other.

The book is quite free from any "padding" and most structures can be rapidly depicted on the surface with accuracy. It will also be found that the author's method is easier of memorization than that of most writers on this subject. The chapter on cranio-cerebral topography is probably the best in the book and is original in many details. The middle meningeal artery, the lateral sinus, the motor and sensory areas and the Sylvian point can be easily and rapidly detected on the operating table. The sections on the side of the face and neck and palm of the hand are also concise and accurate.

The work is finely illustrated and well printed on good quality paper. Altogether it is a useful hand book for student and practitioner.

THE EVOLUTION OF PUBLIC HEALTH NURSING.

IN "The Nurse in Public Health" Mary Beard recounts the evolution of public health nursing from the district nurse of England to the public health nurse with the degree of Bachelor of Nursing of the Yale School of Nursing.[3] Public health nursing in France, Poland, Yugoslavia and Denmark are also reviewed.

In the chapter on "Administration of Midwifery in England" Miss Beard remarks that the maternal mortality rate in England is three and a fraction per thousand births. In the United States of America and Canada it is six and a fraction per thousand births. In Great Britain maternal care has become a primary concern of the Public Health Service. In Denmark, where rational instruction for midwives was established in 1787, the standard of training and education is high. The maternal death rate is only 1·75‰. Danish doctors recognize that midwifery in a normal confinement is as much a nursing function as giving a bath to an enteric fever patient or assisting in the dressing of a wound.

Nursing has passed through two distinct phases in the past fifty years and is now in the third stage. Service, association and now education are the terms that may serve to characterize these phases. In the first period, that of service, Florence Nightingale was under the necessity of reforming many details of hospital management. If details were to receive proper attention the nurses would have to take over the responsibility. It followed that manual service became incorporated in the nurses' training everywhere—she scrubbed and scoured—not because Florence Nightingale believed such tasks essential to her training, but because in the beginning it was necessary to ask the nurses to do them in order to get them done at all.

This book is exceedingly interesting and is full of valuable information not only for nurses, but also for anyone connected with public health work.

INCOMPATIBILITY IN PRESCRIBING.

DR. THOMAS STEPHENSON'S useful little book on incompatibility in prescriptions has met such a demand that a further edition was warranted and has now been published.[1] The first edition appeared in 1915 and the second edition in 1924. The present edition is practically a new book. The work is divided into two parts. The first deals with the general principles of incompatibility. The arrangement is logical and the subject matter extremely clear and well presented. The first section deals with chemical incompatibility and treats of insoluble compounds, evolution of gas, acids and alkalis, decomposition, alkaloids, undesirable compounds. Explosive reactions are admirably presented. Many actual catastrophes are narrated. For instance, the presence of potassium chlorate tablets in the same pocket as lucifer matches has set the clothes on fire. A concentrated solution of potassium chlorate, tincture of perchloride of iron and glycerin is very liable to explode in the warm atmosphere of a sick room. A mixture containing spirit of nitrous ether and almond mixture has been known to explode, probably from the presence of tannin. Hypophosphites are liable to explode when simply heated or triturated in a mortar. Iodine forms explosive compounds with essential oils. With ammonia it forms a very dangerous explosive-nitrogen iodide. The same dangerous body is formed if tincture of iodine be added to compound camphor liniment. Many other disconcerting possibilities are indicated, possibly the most unexpected being a dangerously explosive combination when glycothymoline and solution of hydrogen peroxide are added to water. Section II is devoted to physical or pharmaceutical incompatibility, insolubility, immiscibility, change of colour, consistence and bulk and molecular change. Section III treats of therapeutic incompatibility and very curious and interesting examples are quoted. A patient's face was stippled with black dots of mercury sulphide as the result of a mercurial application followed by a sulphur preparation. The same result may follow the internal administration of mercurials coincidently with the application of sulphur and *vice versa*. The danger is insisted on of giving iodides internally at the same time as mercurials are applied externally (especially to the conjunctiva). Intensely irritating iodide of mercury is formed. Also a case is recorded of a lad taking ichthyol internally and using externally "lactate of lead" lotion. The skin was blackened through the formation of sulphide of lead. Part II contains a dictionary of incompatibles—an alphabetical summary of drugs with their doses, solubilities and incompatibilities. An adequate general index completes this most interesting and useful little work.

---

[1] "Physical Therapeutic Technic," by Frank Butler Granger, A.B., M.D., with a foreword by William D. McFee, M.D.; 1929. Philadelphia: W. B. Sanders Company; Melbourne: James Little. Royal 8vo., pp. 417, with illustrations. Price: 32s. 6d. net.

[2] "Landmarks and Surface Markings of the Human Body," by L. Bathe Rawling, M.B., B.C. (Cant.), F.R.C.S.; Seventh Edition; 1929. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 106, with illustrations. Price: 6s. net.

[3] "The Nurse in Public Health," by Mary Beard; 1929. New York: Harper and Brothers; Crown 8vo., pp. 227. Price: $3.50 net.

[1] "Incompatibility in Prescriptions and How to Avoid it to which is Added a Dictionary of Incompatibilities," by Thomas Stephenson, D.Sc., Ph.C., F.R.S. (Edinburgh), F.C.S.; New Edition, Revised and Enlarged; 1929. Edinburgh: "The Prescriber" Offices. Demy 8vo., pp. 61. Price: 4s. 6d. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 14, 1929.

Going Back to School.

THE undergraduate of medicine often looks forward to the day when on receiving his degree he ceases to be a student. He soon learns his mistake. It is true that he is no longer required to give his whole time and energy to class work, but it is equally true that he has to study from his patients, from journals and from books in order that he may have some understanding of the pathological processes existing in his patient's body. Although the knowledge of the processes going on in health and in disease possessed at the present time is small when compared with the full understanding of these phenomena, a vast amount of information has been accumulated and no one man can master it all. The practitioner of medicine remains a student until the end of his days, struggling to gain a clear insight into the mysteries called life, health, disease and death. The British Medical Association exists primarily for the purpose of promoting medical knowledge and disseminating it. Unfortunately post-graduate education tends to be discontinuous and lacking in system. It is relatively unusual for a medical practitioner to employ the current medical journals, books and reports in such a manner that he is able to study each chapter of medical science systematically and to bring his knowledge stage by stage up to date. He receives a certain amount of assistance by the selection of material and by the collective arrangement of subjects in his journals, but even with this aid he has to seek in many places before he can hope to collect a sufficient store of facts.

An expedient devised to overcome the difficulties inherent in self-education is known as post-graduate study for want of a better term. Post-graduate courses serve an excellent purpose since the teaching is systematized by those who hold hospital appointments. They have continuous access to patients whose affections can be investigated by teams of specialists within the institutions. The fundamental principle on which this movement is based, is that knowledge is best acquired when it has to be imparted to others. Hospital physicians, surgeons or specialists have to inform themselves concerning the work carried out elsewhere and to marshal all the available facts before they essay to teach. They are not restricted as are the teachers of undergraduates, to stereotype questions and answers formulated for examination purposes. They can use their critical judgement and can ignore text book doctrines, if there is sufficient evidence to justify a challenge of their soundness.

In the past this form of post-graduate work has been well organized in Melbourne by the Melbourne Permanent Committee for Post-Graduate Work. Some attempts have been made in Sydney to institute useful courses, but the schemes on which the courses were planned, were defective. More recently the New South Wales Branch of the British Medical Association has created a Post-Graduate Work Committee. The committee consists of the *ex officio* members, the President, the Honorary Secretary, the General Medical Secretary, the Honorary Treasurer and the Honorary Secretary of the Science and Oganization Committee, three members appointed by the Council, Dr. E. H. M. Stephen, Dr. W. W. Ingram and Dr. V. M. Coppleson. Dr. Coppleson was chosen to be the honorary secretary of the committee. Dr. Ingram resigned a short time ago when he joined the Mawson Antarctica expedition. The committee coopted Dr. Wilfred Evans to fill the vacancy and added Dr. A. S. Walker. The cooperation of the University of Sydney and of the members of the staffs of the several metropolitan public hospitals was sought and obtained. It is proposed to hold annual revision courses during the university vacation, courses in special subjects and advanced courses and special lectures. The courses will be open to all registered medical practitioners who make application to be admitted. The first course will probably be held at the end of January, 1930. Provisional arrangements are being made with the large teaching hospitals in Sydney and with certain

special institutions and it is anticipated that the members of the honorary medical staffs will make adequate arrangements and undergo sufficient preparation to insure success of the course. We learn that the revision courses will last a fortnight. Of the working days a certain number will be allotted to the three great metropolitan public hospitals and a shorter period to each of the special institutions. The organization is proceeding satisfactorily and there is reason to anticipate that the committee will work on a concrete plan which will be carried out in its entirety. As long as the individual lecturers and demonstrators are permitted to transform the scheme into a thing of shreds and patches, efficiency is unlikely to be achieved. On the other hand, strong guidance and direction by one or two who have devised the organization, together with loyal cooperation by the individual lecturers and demonstrators, will spell success.

There is room for two good post-graduate work organizations in Australia. The more satisfactory the two become, the closer will be their coordination and the greater will be the value of the liaison. The medical profession has given evidence that it is prepared to provide substantial sums of money for sound courses and special lectures, but it will not waste money on ineffectual attempts. We feel convinced that the new move in Sydney will be well founded and that on this firm basis a substantial superstructure will eventually be erected.

Current Comment.

POLIOMYELITIS.

THE papers by Dr. Jean Macnamara and Dr. Burnet published in this issue were among the most important papers presented at the third session of the Australasian Medical Congress (British Medical Association). They have been published in THE MEDICAL JOURNAL OF AUSTRALIA by special arrangement with the Executive Committee of Congress. The importance of the study of poliomyelitis has long been recognized and the work reported by Dr. Jean Macnamara and Dr. Burnet is the result of concentrated effort extending over several years. Dr. Macnamara's first contribution to the subject was made in our issue of September 1, 1928, as part of a symposium on poliomyelitis. She has given an ample and useful description of the value of human immune serum in treatment and detailed directions for its administration. The first conclusion to be gathered from reading her article is that a knowledge of the histological changes produced by the disease is essential. In the symposium of last year an excellent account of these changes was given by Reginald Webster. At the present time it will be useful if these changes are referred to again and with this object attention may be drawn to some work by E. W. Hurst emanating from the Lister Institute, London.[1]

The tissue studied was obtained by the intracerebral inoculation of 0·5 cubic centimetre of a 5% virulent cord emulsion into eighteen monkeys, *Macacus rhesus*. At the autopsies the usual finding of intense congestion of the meningeal vessels and of the grey matter was noted; this was especially pronounced in the regions of the anterior horns of the spinal cord. On section the nervous tissue was seen to be moister than normal and the cut surface was glistening. Hurst describes his histological findings according to two stages, the stage of advancing paralysis and the stage of recovery. Only one animal survived the acute attack and its nervous system was examined after it had been killed. Although the lesions in the stage of advancing paralysis were most pronounced in the lumbar and cervical enlargements, no level escaped damage and the essential nature of the changes was similar in all regions. In the cord proper vascular congestion was prominent; the cellular lesions are described under three headings, perivascular and extraadventitial infiltration, diffuse and focal infiltration of the nervous tissues and destruction of the nerve cells and their fibres. Perivascular infiltration was usually pronounced both in the larger vessels near the central canal and in the smaller vessels of the anterior and posterior horns. In the earlier stages it was more or less limited to the grey matter, but very soon spread through the white matter to reach the surface of the cord. Perivascular infiltration was not necessarily observed, even in the presence of the most extreme nerve cell destruction; in the most virulent infections it often appeared insignificant when compared with the damage sustained by the neurones. Tissue infiltration appeared as a diffuse cellular increase in the grey matter of the anterior horns, in the grey commissure and in a large part of the posterior horns. Numerous focal accumulations of cells were found. The polymorphonuclear leucocytes varied according to the stage of the disease and the virulence of the virus; they disappeared with remarkable rapidity and forty-eight hours after the appearance of the first nervous symptom few as a rule were found. Lymphocytes were scattered through the tissues and took part in the focal infiltrations; they were more numerous with the less virulent strains of virus and in the later stages of infection. The microglia was found to have undergone enormous proliferation and except when the polymorpho-

[1] *The Journal of Pathology and Bacteriology*, July, 1929.

nuclear invasion was excessive during the first stage, formed the bulk of the exudate of the grey matter. Nerve cell destruction was most pronounced in the cervical and lumbar regions; commonly from one-half to two-thirds of the anterior horn cells were destroyed. Sometimes the destruction was greater than this and sometimes in the dorsal region the destruction was less severe. The damage was not confined to the anterior horn cells. Sometimes the nerve cells could be seen undergoing phagocytosis. With a less virulent infection the sequence of degenerative changes could be more easily followed. In neuronophagia, in addition to polymorphonuclear cells which varied in number, a few lymphocytes and glial cells, microglial elements rapidly gathered and assumed the leading rôle, removing the dead cells with great rapidity. Hurst also describes lesions in the medulla and pons, the mid-brain, the basal ganglia, the cortex, the cerebellum, intervertebral and Gasserian ganglia and in other parts of the body; the latter are those of a general systemic infection. In one monkey which recovered, the histological picture was that of a central nervous system which was normal, except that a large proportion of the nerve cells had been destroyed.

It will be admitted at once that the description given by Hurst contains nothing very new; most of the points brought out by him were mentioned by Webster in his description. The first and possibly the most important fact is that poliomyelitis is not a disease confined to the anterior horn cells. The whole nervous system is affected and in fact the whole body. The brunt of the attack falls on to the anterior horn cells. The reason for the picking out of the anterior horn cells is not clear. In human beings the infection is regarded as occurring from the naso-pharynx and the infective agent is probably carried to the central nervous system by the blood stream. In Hurst's animals the virus was introduced directly into the cerebral tissue. From the cerebral areas it might be carried to the cord either by the cerebro-spinal fluid which is most likely, or by the blood stream. That it spreads by direct extension is unlikely in view of the finding of most pronounced lesions in the lumbar region and in the cervical part of the cord. It should be remembered, as Webster has pointed out, that when the virus has been adapted to monkeys, infection can be caused by the naso-pharyngeal route. As far as the involvement of the anterior horn cells in far greater degree than any other cells is concerned, either the virus must have a special affinity for these cells, a kind of specialized neurotropic quality, or there must be a preliminary making ready of the soil which is to receive the infecting agent. This would explain, at any rate in some instances, the apparent variation in virulence of the infecting agent. The truth probably is that both a neurotropic element and a local change are necessary. Otherwise it would be difficult to explain the picking out of one muscle or set of muscles or the cerebral form which is occasionally seen.

The second point to be gathered from this work is the extreme rapidity with which cell destruction takes place. Macnamara's clinical finding, that only in the preparalytic stage can serum produce a healing effect, is easily understandable. The nerve cell is killed very quickly. When degenerative changes have actually begun, it will be useless to give serum. It is also of interest to consider Macnamara's patients whose infection was of the spreading, ascending type. That the paralysis of some was arrested shows that the serum was able to reach the cells threatened or not seriously affected. The failure to reach these cells in some instances is explained by Macnamara as due to the choking of the vessels, œdema *et cetera*. When serum is introduced into the theca, it is presumably absorbed by the tissues of the anterior horn cells. According to Macnamara's view the changes which she mentions, are sufficient to prevent this absorption and this is apparently a valid explanation.

Among the other aspects discussed by Hurst is that of the relationship of interstitial inflammation to the nerve cell degeneration. As a result of his observations and the findings of others he comes to the conclusion that the nerve cells are primarily affected by the virus and that the degeneration is not due to the accompanying interstitial inflammation. After a careful description of the different cells taking part in the cellular reaction, he refers to the question of the presence of hæmorrhages in the cord in poliomyelitis. He points out that they have been regarded as terminal phenomena due to the asphyxia to which most patients succumb. He agrees with Strauss that if this is so, they should also be found in the serous membranes. In some of his animals killed before the onset of respiratory paralysis, hæmorrhages were present. They were also found in a normal control animal when the precautions against kinking the cord during removal were somewhat relaxed. He thinks that the hæmorrhages are caused by trauma during removal of the cord and he states that the congested nature of the tissues would make rupture of the vessels easier to produce. It would be worth while attempting to harden a cord *in situ* to determine this point.

THE USE OF GLYCERIN IN CÆSAREAN SECTION.

THE good results obtained by the use of glycerin in puerperal sepsis have prompted Dr. Margaret Salmond, on the advice of Professor Louise McIlroy, of the Royal Free Hospital, London, to adopt it in Cæsarean section.[1] After the removal of the placenta the uterine cavity is swabbed out with gauze dipped in glycerin and held on a sponge holder. The uterus retracts when it is wrapped in hot towels. It is held that by this means hysterectomy will often be avoided and risk of sepsis lessened. The method is worthy of trial.

[1] *The Lancet*, September 28, 1929.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Nerves in Cancers.

HORST OERTEL (*The Journal of Pathology and Bacteriology*, July, 1929) has investigated the innervation of human cancers. The freezing method of cutting sections was used and sections were between 25 and 30 microns thick. After mordanting with pyridin the sections were treated alternately with an ammoniacal silver bath, distilled water, formalin, distilled water, cyanide of potash, distilled water, chloride of gold and acetic acid, water, sodium thiosulphate, water, hæmatoxylin, absolute alcohol. The nerves were identified by the selective action of the stain, the arrangement, distribution and manner of branching and the formal character of the fibres themselves and their endings. The essential point was to determine whether the nerves were part of the tumour growth or whether they were remaining tissue nerves in the tumour field. The author points out that the sections of which he reproduces illustrations, were obtained from the main mass of a well established tumour. The view that they are remnants can be excluded by their location and by the fact that they form a delicate connected system with equally definite relations to the tumour parenchyma and stroma. The nerve supply is a twofold one, similar to that of any other glandular tissue. Nerves appear in abundance of bundles and fibres in stroma where they follow fibres and vessels. Nerve fibres run across the field with a vascular spiral. In the stroma characteristic delicate plexuses are present. From the plexuses nerve fibrils approach and are intimately attached to cancer nests which they encircle; fibres penetrate the parenchyma. The author concludes that the nervous organization is an integral part of the cancer growth. He concludes with the observation that in the tumour and cancer problem attention has been focused too strongly on the cells and not sufficiently on the environmental influences of blood vessels and nerves.

Hæmangioma of Bone.

PAUL C. BUCY (*The American Journal of Pathology*, July, 1929) discusses the pathology of hæmangioma of bone. He bases his description on seven cases. The tissue is generally composed of large cavernous spaces which contain blood cells and are lined with a layer of flat endothelium. The intravascular tissue is loose and rather acellular. The cells are usually spindle shaped, but some are stellate. The stroma of the connective tissue consists of a loose network of fibres varying greatly in amount. In one of the tumours there were found cells with a small, rather compact, deeply staining nucleus with a large rounded cytoplasm containing granules. These were regarded as true myelocytes. As a rule the vascular spaces are lined by a single layer of flattened endothelial cells. Occasionally places were seen in which the lining consisted of two or three layers of cells. The vessels usually consist of large cavernous spaces. In one instance the lumina of the vessels corresponded in size to those of capillaries. Microscopical examination of the bony trabeculæ revealed two changes, degenerative and formative. The degenerative changes are manifest by necrosis and absorption. The formative changes are more commonly evidenced by spicules of newly formed bone about whose surface can be seen a closely packed layer of osteoblasts. Less commonly there is a transformation of fibro-cartilage into bone. The authors state that there is nothing in their experience to throw any light upon the ætiology of these tumours. They are certainly benign and no microscopical evidence of malignancy can be discovered. There is no reason to conclude that the destructive and formative changes in the bone are a primary part of the neoplastic process, but there is every reason to believe that they are only secondary reactive phenomena.

The Skull in Osteosclerotic Anæmia.

DAVID M. GREIG (*Edinburgh Medical Journal*, August, 1929) states that the two prominent features of osteosclerotic anæmia are the patchy or generalized sclerosis of long bones and the anæmia. There is a form of osteosclerotic anæmia which is congenital in origin; possibly there is a form which may be acquired late in life and it may be familial and hereditary. The author distinguishes between the generalized affections in which the primary disease lies in the hæmatopoietic organs, and true osteosclerotic anæmia in which the first obvious change occurs in the bones and the anæmia is secondary. He describes in detail the skull of a boy, aged eleven years, who suffered from osteosclerotic anæmia. The appearance of the skull gave an impression of solidity and compactness. The bones of the face and cranium were dense and eburnated. Most of the sutures had undergone synostosis. In osteosclerotic anæmia the skull changes are congenital and there is a secondary interference with the development of the brain. The author refers to the statements in the literature that the base of the skull is found to be thickened on radiography. He points out that there is increased density, but that this does not necessarily mean that thickening is present. He shows that in the skull described by him calvarium was thickened and that the diploe had undergone sclerosis. It is often stated in the literature that there is a frequent occurrence of spontaneous and multiple or single and recurrent fractures in this condition. The author holds that typical osteosclerotic anæmia can occur in children without fractures and that it is wrong to regard the condition as synonymous with *osteosclerosis fragilitas generalisata* as is done by certain authors. The belief that fractures are a characteristic of osteosclerotic anæmia is partly responsible for the confusion which surrounds the condition.

The Diameters of Erythrocytes.

C. PRICE-JONES (*The Journal of Pathology and Bacteriology*, July, 1929) has investigated the diameters of erythrocytes in normal persons and persons suffering from pernicious anæmia. The healthy persons numbered one hundred, and five hundred cells were measured in each instance. The normal persons were not selected and varied in age from nineteen to ninety years. The author used the dry film method. He found that the average mean diameter was 7·202 μ, the healthy range being from 6·686 μ to 7·718 μ. The healthy range of coefficients of variation was from 5·33 to 7·32 with a mean of 6·33. An excessive variability was found to be more constant in pernicious anæmia than a high mean diameter. The effect of liver treatment was followed in twelve patients. In eleven instances the mean diameter became smaller and in nine it returned to within the normal range. In three instances the diameter differed from the healthy mean by less than the standard deviation and in three by less than twice the standard deviation. In only one instance and on one occasion was the diameter brought to or to the left of the healthy mean. The author points out that if the erythrocytes were completely restored to a healthy condition and if pernicious anæmia occurred in persons whose mean erythrocyte diameters before the onset of the disease were distributed in a normal fashion, it would be expected that at least in some of the nine cases the diameters after treatment would be at or below the healthy mean. Their actual distribution was just compatible with the chances of random sampling of a normal distribution, but the author thinks that it is unlikely that it is wholly due to this. The failure to reach the healthy mean was remarkable in three patients who before treatment had mean diameters within the healthy range.

Pleural Neoplasms.

E. KUX (*Virchow's Archiv für Pathologische Anatomie und Physiologie und für Klinische Medizin*, June, 1929) discusses primary growths of the pleura. He describes in detail three tumours, clinical histories and pathological findings. In all three instances the growth involved the parietal and visceral pleura and the regional lymphatic glands. The structure of two of the tumours was that of a carcinoma and of the third tumour that of a sarcoma. The author refers to the recent work of Maximow in tumour culture experiments and to his finding that superficial pleural cells can produce either connective tissue cells or epithelial cells. He points out that in the three tumours

described by him, the structure was neither purely carcinomatous nor sarcomatous, but that one or other type predominated. He holds that they demonstrate the fact that several possibilities or potentialities for growth reside in the superficial cells of the pleura and in all serous layers. The superficial cells of the pleura cannot be regarded as endothelial and the use of the term endothelioma should be confined to those tumours which actually originate in endothelium arising from the mesenchyme and lining the lymph and blood spaces. To describe growths of the serous membranes as pleuromata, pericardiomata and so forth would only add to the already long list of names. Until more definite knowledge of the superficial cells is obtained, it is better to describe these tumours as superficial cell tumours or mesotheliomata.

Mitoses in Normal and Malignant Tissues.

ALBERT FISCHER AND RAYMOND C. PARKER (*The British Journal of Experimental Pathology*, October, 1929) have studied mitosis in normal and malignant tissue. The normal tissue examined by them consisted of fibroblasts in the eighth passage removed from the zone of outgrowth of the periosteum of bone of a sixteen day old chick embryo. The malignant tissue was obtained from a forty-eight hour old cover glass culture of Ehrlich adenocarcinoma of a mouse. The cultures were fixed and cut in serial sections. The authors found that the percentage of cells in mitosis in malignant tissue greatly exceeded the corresponding percentage in normal tissue, in spite of the fact that the growth rate of normal tissue may be several hundred times that of malignant tissue. In cultures of normal tissue the greatest number of dividing cells is to be found in the marginal areas. In cultures of malignant tissue there is no appreciable difference between the number of dividing cells to be found in the central and marginal areas. The authors explain their findings by suggesting that the average duration of life of the carcinoma cell must be extremely short. A large percentage of cells dies at every moment. These losses are compensated by the regeneration of new cells. When the generation of new cells exceeds the loss through death, growth results. Growth in a carcinoma culture is therefore not a necessary result of cell division, but is rather an index of the potencies of the component cells to initiate cell regeneration without the intervention of external stimuli.

MORPHOLOGY.

The Structure of Cells in Tissues.

GEORGE ST. DE RÉNYI (*The Journal of Comparative Neurology*, April 15, 1929) has investigated the physical properties of the living axis cylinder in the myelinated nerve fibre of the frog. He carried out his observations by microdissection. He found that the cross sectional areas of the axis cylinder and the myelin sheath of the normal living frog nerve fibres are related to one another in the ratio of one to one. If the measurement shows a different relationship, the nerve fibre cannot be regarded as normal. The Henle sheath is adherent to the nerve fibre, but is not inseparable from it. This sheath sticks to the working instruments and has a definite fibrillar structure. The fibrils of Henle's sheath are of elastic nature. The axis cylinder really possesses its original shape. Its substance is of soft jelly-like consistency, plastic and elastic to a certain extent. Neurilemma and myelin do not manifest elastic properties. The author found no difference between the surface and the interior of the axone as far as their physical state is concerned. The substance of the node is more viscous and solid than the structure of the internode. Normal living axones do not manifest neurofibrillar structure.

Growth of the Human Stomach.

G. H. SCOTT (*The Anatomical Record*, July 25, 1929) has investigated the growth in surface area of the human gastric mucosa. He bases his conclusions on observations of ninety-six stomachs from individuals ranging in age from six fœtal months to fifty years. The method which is described in detail, included hardening, the making of a plaster cast, the fitting of tissue paper to the cast, the cutting out of a pattern of the tissue paper on standard paper and the calculation of the area by estimating the weight of the latter paper. The author claims from control observations that the method is reliable. He found that the mucosal area increases 54·46 fold between the sixth fœtal month and maturity and 13·42 fold from birth to the adult state. The most rapid growth takes place in the first three months of postnatal life; during this time it increases over 100% per month. Normal distension has little or no effect upon the amount of surface presented to the gastric cavity. Distension under pressures near to the bursting strength results in an extension of the area of the mucosa; this extension is evidently brought about by shortening of the crypts and glands. No change takes place in the shape of the epithelial cells themselves.

G. H. SCOTT (*The American Journal of Anatomy*, July 15, 1929) has made a quantitative study of the fœtal growth changes in the parts of the human stomach wall. The material studied consisted of forty-five fœtuses and new-born infants, varying in age from 5·4 fœtal months to birth. He concludes that the relative amount of surface epithelium seen in a cross section area of the stomach wall does not change materially. The computed weight of the surface epithelium per square centimetre of gastric wall increases steadily until the last month of fœtal life. There is a slight decrease in this value at birth. The calculated weight of the surface epithelium for the entire stomach increases steadily until the time of birth. The relative amount of crypt epithelium increases steadily until the last fœtal month. By the time of birth the relative amount of crypt epithelium has undergone a slight decrease. During the last month of fœtal life there is a drop of about one-third in the computed weight of crypt epithelium of the entire stomach. The relative amount of gland substance increases until the last month of fœtal life. In the last month of fœtal life and at birth there is a progressive decrease in the relative value of the gland substance. At birth the glands form over 30% of the gastric mucosa. The computed weight of the gastric glands follows the same change as the crypt epithelium. The relative amount of the remainder of the mucosa as seen in a cross section of the stomach wall, increases steadily until the close of the ninth fœtal month. In the last prenatal month and at birth the percentage of the remainder of the mucosa decreases. The computed weight of the remainder of the mucosa per square centimetre of stomach wall increases steadily until the tenth fœtal month. This value remains practically constant. The calculated weight of the remainder of the mucosa in the entire stomach increases steadily with the advance in fœtal age. The relative amount of submucosa as seen in a cross section area of the stomach wall fluctuates considerably until the time of birth. At birth the submucosa forms nearly one-third of the gastric wall.

The Olfactory Striæ in Man.

MAX KLASS (*The Journal of Comparative Neurology*, February 15, 1929) has made a study of the olfactory striation in thirty-five hemispheres from human subjects other than Europeans. He concludes that the fundamental pattern of double components, as first discovered and recorded by Baccari and subsequently investigated in detail by Mutel, is genuine. The only standard strial formation with which all brains may be compared, is the one in which all the striæ are present, that is the "four-strial" type. The four-strial as well as the two-strial formation does not occur so often as the three-strial formation. The "A" type of formation dominates all the rest and was found to be present in 55% of the material examined. It is present in both European and Bantu brains. The incidence of the lateral accessory stria preponderates over that of the medial accessory stria. No group or race has a strial pattern peculiar to itself. When the olfactory tracts are divided into their component elements, the medial and lateral striæ are invariably found to be present. There is nothing in the strial formation of European, Bantu, Eurafrican or Bushnegroid brains which enables them to be distinguished from one another.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Sydney Hospital on July 11, 1929. The meeting took the form of a series of demonstrations by members of the honorary staff.

Familial Acholuric Jaundice.

DR. G. C. WILLCOCKS showed a female child, aged eleven years. One month previously the child had suffered from an attack of jaundice and soon afterwards had felt sick and feverish and had vomited. She had had no pain and no headache, but had had three or four similar attacks during the last two years, the attacks lasting for from three to four days. The family history was of some interest. The father, aged forty-two years, suffered from Parkinson's disease; the mother, aged thirty-eight, had been jaundiced since the birth of the first child. She was subject to bilious attacks, headaches, vomiting and epigastric pain. She had had six children. The eldest child, a girl, aged seventeen, had been jaundiced when a child and again at fourteen years of age. The jaundice had persisted since that time. She suffered from attacks of vomiting, headache and epigastric pain. The second child, a male, had died at the age of ten as the result of an accident, but he had also suffered from jaundice. The third child, a girl, aged thirteen years, had been jaundiced since she was six and a half years old. She suffered from attacks of headache and pain. The patient was the fourth child. The fifth child, a male, aged eight years, had not suffered from jaundice. The sixth child, aged four years, had been jaundiced at birth and had suffered from one attack since that time. During the patient's attacks the pain was not severe, it lasted in spasms for from one-half to one hour and was not related to meals. The pain which always occurred in the epigastrium, was accompanied by vomiting of bile, by feverishness and by the passage of putty-like stools. The last attack had occurred seven months before the patient's admission. She had been nervous and frightened for the previous two years. She had been treated for thread worms for nine months and the treatment had been stopped three months previously. The urine had sometimes had a reddish appearance and a pinkish sediment. On examination the child was found to be jaundiced. The spleen was palpable. The circulatory system was normal. The systolic blood pressure was 120 and the diastolic pressure 70 millimetres of mercury. No abnormality was discoverable in either the respiratory or the nervous system. Examination of the blood revealed anæmia and increased fragility of the red cells; hæmolysis began at a concentration of 0·5% saline solution and was complete at 0·4%. The Van den Bergh test yielded an indirect positive response. Dr. Willcocks said that jaundice, enlarged spleen, anæmia and increased fragility of the red cells with a family history of jaundice were typical of familial or congenital acholuric jaundice. The cause was unknown. Removal of the spleen cured the condition, but this was done only if the symptoms were severe.

Congenital Syphilis.

DR. WILFRED EVANS showed two sisters, aged fourteen and seventeen years respectively, who were suffering from interstitial keratitis and who manifested other stigmata of congenital syphilis. The mother gave a history of several miscarriages in the later months of pregnancy, followed by the birth of these two girls at term. The younger sister was a well nourished girl, but her face presented characteristic features of congenital syphilis. Both frontal bones were bossy, interstitial keratitis was present in the left eye, a large puckered scar was visible on the upper lip, involving the lower part of the *septum nasi* and the upper left medial incisor tooth was peg shaped and had a concave longer margin, as described by Hutchinson. An interesting feature was that she had first come for treatment three years previously on account of a gumma of the external nares and two years later after a long period of intensive treatment had developd interstitial keratitis of the left eye. The elder sister presented only two features of interest, an extensive periosteal thickening of the left tibia with early sabre blade deformity and a recent acute interstitial keratitis of the left eye.

Enlargement of the Legs of Unknown Causation.

DR. E. H. STOKES showed a woman, forty-nine years of age, who was married and had five children. She had suffered from enlargement of the legs for three years. The swelling commenced on the dorsum of the foot and extended up to but not above the knee joint. The ankle joint was clearly demarcated and in this respect the condition resembled Milroy's disease. The right leg was affected to a far greater degree than the left. No other members of the family suffered from a similar disability. The enlargement was of the nature of a hyperplasia rather than an œdema. The patient also had a large uterine fibromyoma and had at times suffered from acute retention of urine. Early in May, 1929, her basal metabolic rate had been −6%. She had manifested no improvement as a result of treatment with thyreoid extract. There was no evidence of cardiac or renal disease. The cause of the enlargement was as yet unknown, but did not appear to be associated with the uterine tumour.

Tetany.

Dr. Stokes also showed a woman, aged twenty-nine years, who had been subjected to thyreoidectomy six and a half years previously. The day following the operation she had noticed spasms in all the muscles of the body. The hands and feet had been especially affected. During the attacks the arms had been adducted, the forearms flexed and the hands drawn into a cone shape. The legs had been flexed at the knees and the toes drawn into the soles of the feet. The spasms had usually persisted for about a week. The patient had often had about a week's freedom from the attacks. She was married and had a child eight months of age. It was interesting to note that for a whole year, while the patient was pregnant and for three months afterwards, the attacks had ceased. At the time of the meeting Trousseau's phenomenon and Chvostek's phenomenon could be demonstrated. The muscles had also exhibited increased excitability to electrical stimulation (Erb's reaction). The calcium content of the blood had been estimated from time to time. It had been found to be lower than normal. On January 23, 1929, it had been 9·6 milligrammes per hundred cubic centimetres. On January 29 the patient had had a severe attack and the calcium content had been 8·5 milligrammes per hundred cubic centimetres. The lowest reading had been 6·3 milligrammes. The patient had been treated by calcium lactate given in doses of 1·2 grammes (twenty grains) by mouth three times a day after food. "Paroidin" had been injected hypodermically in doses of 0·2 mil twice a day during alternate weeks. The patient had received this treatment for four months and had received considerable benefit.

Lipoid Nephrosis.

The next patient shown by Dr. Stokes was a young man, aged seventeen years. About nine months previously he had suffered from pleurisy and pneumonia. Shortly after this he had noticed that his face was swollen and his eyelids were puffy. On admission to hospital three months before the meeting his pallor had been evident; puffiness of the eyelids, œdema of the feet, ankles, scrotum and penis had been noted. Only a small amount of urine had been voided during the two days prior to admission. The abdomen had been swollen, but no definite signs of free fluid had been found. The heart had been normal in size. The urine had been acid in reaction, had had a specific gravity of 1020 and had contained a large amount of albumin, but no sugar. The blood pressure had been normal. The blood sodium chloride had been 520 milligrammes per hundred cubic centimetres. The basal metabolic rate had been −17%. The blood urea nitrogen had been twenty-six milligrammes per hundred cubic

centimetres, the blood creatinin two milligrammes, the blood non-protein nitrogen forty-one milligrammes and the blood serum cholesterol eight hundred milligrammes. The blood count had been normal. The blood serum had not reacted to the Wassermann test. Microscopical examination of the urine had revealed a few pus cells and red cells and granular casts in abundance. The *fundi oculorum* had been found to be normal. On April 24, 1929, the patient had been given thyreoid extract 0·06 gramme (one grain) three times a day. The dose had been gradually increased and at the time of the meeting the patient was taking 0·48 gramme (eight grains) three times a day. On May 20, 1929, the patient had suffered from an attack of bronchopneumonia. He had recovered from this. For the previous six weeks he had been taking a high protein diet with fifteen grammes of urea twice a day. On July 4, 1929, the basal metabolic rate had been −12%, the blood urea nitrogen had been thirty-two milligrammes per hundred cubic centimetres, the blood creatinin two milligrammes and the blood serum cholesterol 615 milligrammes. The average amount of urine passed every day was about two litres (seventy fluid ounces). The systolic blood pressure was 162 and the diastolic pressure 88 millimetres of mercury. The highest blood serum cholesterol reading recorded had been 842 milligrammes per hundred cubic centimetres. Dr. Stokes said that the patient was probably suffering from lipoid nephrosis in combination with diffuse glomerulonephritis (parenchymatous nephritis). The œdema, the albuminuria, the high blood cholesterol and the low metabolic rate were characteristic of lipoid nephrosis, while the cellular nature of the urinary deposit, the rising blood pressure and the increasing blood urea nitrogen suggested the addition of nephritis.

Psycho-Galvanic Reflex and Psychogalvanometer.

DR. R. C. WINN showed several patients in order to illustrate the importance of the psycho-galvanic reflex and the practicability of the psychogalvanometer.

The demonstration was carried out by a moving picture film.

A male patient, aged seventeen years, was in hospital for operative treatment and the demonstration was made by courtesy of Dr. George Bell. On this subject were demonstrated normal psycho-galvanic reflexes to a variety of stimuli. The psycho-galvanic reflex was the name given to the diminution in resistance of the skin to the passage of an electric current that was produced by emotion. Any emotional stimulus such as the sound of an unexpected clap of the hands would be followed by a movement of the galvanometer needle. The first stimulus used was a pin prick and this was followed by a swing of the needle to the limit of the dial on the galvanometer. If organic anæsthesia had been present, there would have been no response. Hysterical anæsthesia could usually be differentiated with the psychogalvanometer from assumed anæsthesia by appropriate methods.

The next stimulus used was an unexpected sound and this also produced a very pronounced swing of the galvanometer needle. It was pointed out that if organic deafness had been present, there would have been no response and that hysterical deafness and malingering could usually be detected by this reflex. This was followed by the demonstration of the psycho-galvanic reflex following a threatening movement to the eyes; it was obvious that organic blindness, hysterical blindness and assumed blindness could be tested in the same way as anæsthesia and deafness.

The reflex following forcible hyperflexions of the finger (such as school boys delight in inflicting on their fellows) was then shown. It was pointed out that any painful movement (such as that of a rheumatic joint) or painful pressure (such as that over a tender muscle) would give a psycho-galvanic reflex. In consequence it was possible to obtain objective evidence of pain in such conditions.

The psycho-galvanic response to word stimulus was brought about by the investigator saying a word and the subject responding with the first word that came to his mind. All words gave a reflex response, probably because of the mental effort, but a word having an emotional association, such as "operation," gave a more pronounced reaction.

It was indicated that word stimulus could be used in psychotherapy and that during analysis of a neurotic patient's syndrome the phycho-galvanic response would give invaluable help as an indicator of emotional states through which the patient was passing.

The second patient, a male, aged forty-four years, was suffering from an incised wound of the thigh and was shown by courtesy of Dr. Ramsay Sharp. It was intended to demonstrate that a patient with complete anæsthesia would have no psycho-galvanic reflex following pricking of the anæsthetic area with a pin or following deep pressure in the same area. As no patient was available with complete anæsthesia, this patient was shown. He had a narrow area of hypoæsthesia extending a few centimetres below the wound. It was demonstrated that pin prick stimulation of this area was followed by very much reduced excursions of the galvanometer needle compared with stimulation of any other area. In a test done on July 11, 1929, the average deflection of the hypoæsthetic area was eighteen divisions of the dial and of normal skin twenty-eight.

The third patient was a female, aged thirty-seven years, suffering from anxiety hysteria. She had been referred by Dr. Lyle Buchanan. This patient complained of a circular area of anæsthesia extending five centimetres (two inches) from the margin of the patella which she had had since the aspiration of a prepatellar bursa. She always gave a psycho-galvanic reflex following stimulation of the "anæsthetic" area by pricking with a pin and in consequence an organic lesion was excluded. This occurred despite the fact that owing to a reaction to the Wassermann test *tabes dorsalis* had previously been a possibility. The differential diagnosis was thus reduced to three conditions, hysteria (pure), anxiety hysteria, malingering. According to Golla, hysteria gave a diminished psycho-galvanic reflex following all forms of stimulation; as this patient gave exaggerated responses she could not be suffering from pure hysteria, if Golla's findings were correct. Exaggerated psycho-galvanic reflexes occurred in anxiety states; consequently a diagnosis of anxiety state combined with hysteria would be in keeping with the facts unless she happened to be a malingerer. Malingering was elminated by using word stimulus; the words "imposter" and "fraud" were included in the series of words used and the variations in psycho-galvanic response were noted; these and the nature of the patient's reaction to inquiry were such as to convince the investigator that she was not malingering. This patient illustrated the value of the psycho-galvanic reflex as an aid in diagnosis.

Ununited Fracture of the Femur.

DR. GEORGE BELL showed a male patient, aged forty-one years, who had been admitted to hospital on June 26, 1929. Eight months previously the patient had been sitting in a chair with his legs crossed. He had thought that the chair was going to fall and in uncrossing his legs had broken his right femur. He had been in a hospital in the country ever since. He had been placed in a frame for five weeks and since then had been in a Thomas's splint. There was a fracture of the femur about its middle and but little union was present. There was shortening to the extent of 6·25 centimetres. X ray examination revealed no union.

Fracture of the Patella.

Dr. Bell's second patient was a young man, aged eighteen years, who had been admitted to hospital on May 21, 1929, with a fractured patella. He had been playing football and while jumping into the air to catch the ball, he had heard a crack in his right knee. He had been completely off the ground when it happened. He had kept on playing for fifteen minutes, but had finally stopped and had gone home. He had seen a doctor who made a diagnosis of sprain. On the following day the knee had still been painful and on X ray examination a fracture of the patella had been discovered. The report had been to the effect that there was a transverse fracture of the patella with no separation of the fragments. There had been

slight tenderness over the patella. The patient had been under treatment for gastric ulcer. On May 25, 1929, his leg had been placed on a back splint made of gouching. X ray examination had shown that the fragments were in a satisfactory position. The patient had been discharged with the limb in plaster. The plaster had been removed on July 9, 1929.

Dr. Bell's next patient was a man, aged twenty-three years, who had been admitted to hospital on June 1, 1929, with a history of having fallen from a train. He had had a comminuted fracture of the left patella into the knee joint. There had also been a superficial lacerated wound of the right knee. Operation had been undertaken four hours after admission. Under general anæsthesia the skin had been shaved and cleansed with ether. The wound in the skin had been excised and the patella examined. The median portion had been broken into small, loose fragments. It had been cleansed with ether. The loose fragments had been removed and about the lateral half of the patella only had been left. The knee joint had been swabbed well with ether and closed. Then bismuth, iodoform and paraffin paste had been rubbed into the tissues and the superficial tissues had been brought together loosely with interrupted sutures of silkworm gut. The highest temperature registered during convalescence had been 37·9° C. (100·3° F.) and the highest pulse rate 138. There had been no signs of acute inflammation, but some sero-pus mixed with the bismuth, iodoform and paraffin paste had been discharged through the superficial part of the wound.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Hill, Henry Manners, M.B., B.S., 1928 (Univ. Melbourne), Alfred Hospital, Prahran.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Magill, William Louis, M.B., B.S., 1927. (Univ. Sydney), "Des-Den," Old South Head Road, Rose Bay.

Medical Societies.

THE BRISBANE HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE BRISBANE HOSPITAL CLINICAL SOCIETY was held at the Brisbane General Hospital on June 13, 1929, DR. J. M. THOMPSON, the President, in the chair.

Crush Burn of the Hand.

DR. ALLAN E. LEE showed a young woman, seventeen years of age, who had injured her right hand two years previously while working in a laundry. Her hand had been crushed between the rollers of a mangle, one of which was hot and the dorsum, as well as being crushed, had been extensively burned. Her condition after several months in hospital had been as follows. There had been an extensive scar on the dorsum of the hand and fingers which had contracted so that the fingers were dorsiflexed to such an extent that the intact thumb could not oppose any of them. As a result the hand had been useless. In addition, the index and little fingers had been pulled over behind the middle pair and the whole had grown together into a single mass of tissue from which only the distal portions of the fingers as far as the distal interphalangeal joints protruded.

There had been a persistent discharging sinus in the centre of the mass of scar tissue, which X rays had shown to be due to a sequestrum in the proximal phalanx of the middle finger. The proximal interphalangeal joints of the four fingers had been akylosed in a position of moderate flexion.

The following procedures had been carried out on the hand.

Under general anæsthesia, the sequestrum responsible for the discharging sinus had been thoroughly scraped away. Two weeks later, again under general anæsthesia, the whole mass of scar tissue on the dorsum of the hand and fingers had been dissected away and the fingers separated down to their bases. This had necessitated chisselling through areas of bony union between the phalanges. A suitable area of skin in the left lower quadrant of the abdominal wall had then been undermined in such a way that the injured hand had been slipped beneath it and the flap had been made to lie against the rawed area. The skin at the wrist end of this area had been accurately sutured to the right margin of the flap. The whole of the arm and body had finally been enveloped in a plaster of Paris case.

During the following three weeks the pedicles were divided, first on the upper side and the transplanted skin had been moulded as well as possible between the fingers. In spite of the large flap used, there had not been sufficient skin to cover the fingers completely. A further source of difficulty had been the great difference in thickness between the atrophied skin on the fingers and the thick transplanted flap, rendering accurate suturing of the edges impossible.

A vigorous course of massage had then been instituted and within two months the patient had found it possible to carry out every possible action with the hand, although to an onlooker the mobility of the fingers still left very much to be desired. Gradual improvement had taken place during the next few months, but unfortunately the index and middle fingers had become webbed together as far as the proximal interphalangeal joint.

This had been corrected as follows. Under local anæsthesia the fingers had been separated right back to between the knuckles. A U-shaped flap of what had originally been abdominal skin had been dissected up from the dorsum of the hand and turned downwards so as to cover the side of one finger completely and also the angle between them. The side of the other finger had been covered with a Thiersch graft taken from the front of the thigh. This procedure had satisfactorily fulfilled its purpose.

At the time of demonstration the hand was a perfect functioning instrument, even delicate actions like holding a needle being performed with ease. The fingers could be flexed separately to 90° at the metacarpo-phalangeal joints and all other movements were free except at the proximal interphalangeal joints, where ankylosis obtained.

Although the patient had received compensation for a permanent total disablement of the hand, she was again earning her living.

Acute Intestinal Obstruction.

The history of Dr. Lee's second patient was as follows. During the course of his occupation he had been struck by the open door of a moving railway carriage and had been dragged along by it for a short distance. On admission to hospital he had presented no definite evidence of intra-abdominal injury, although the honorary surgeon who saw him shortly afterwards had been rather suspicious that some damage had occurred.

Six days afterwards the patient on attempting to pass urine, had found himself unable to do so. A moment or two after desisting from straining, he had been seized with a sudden acute pain in the lower part of the abdomen which had persisted and had spread over the whole abdomen. When seen a few minutes afterwards, he had been lying with the legs drawn up and an absolutely rigid abdominal wall. The withdrawal of ninety centimetres (three ounces) of clear urine had seemed to negative a rupture of the bladder and a tentative diagnosis of ruptured duodenal ulcer had been made.

Under general anæsthesia a small suprapubic incision had been made and fresh intraperitoneal blood encountered. The incision had been extended upwards till an elastic boggy mass had been felt either under or in the ascending colon. This mass on exposure had proved to be a ruptured right kidney surrounded by an extensive hæmatoma. No actual peritoneal rupture had been found, but this had

manifestly been the source of the intraperitoneal blood. It had not been deemed necessary to remove the kidney and the abdominal incision had been closed. Within three days the patient had obviously been suffering from an acute intestinal obstruction and his condition had been rapidly deteriorating.

Under local anæsthesia a distended loop of ileum had been exposed and a tube sewn into it. This had served to relieve the obstruction and nineteen days later further operation had been undertaken. It had then been found that the obstruction was due to multiple firm adhesions between the distal coils of ileum and the under surface of the primary incision. By good fortune the ileostomy had been performed in the next most proximal coil. The entering loop of the ileostomy had been divided, the lower end closed, while the upper was anastomosed into the side of the transverse colon. Only a little mucus had escaped from the opening and the patient had left hospital reasonably well, except for a hernia of the first incision.

During the next few months considerable pain had been experienced in the right iliac fossa and at one stage a diagnosis of appendiceal abscess had been entertained. A mucopurulent discharge had also begun to appear at two spots along the primary wound. Later intestinal mucous membrane had been visible in the depth of these sinuses and later still a piece of inverted ileum 7·5 centimetres (three inches) long had begun to protrude through one of them. Evidently there had been an inflammatory process of such severity in the coils of obstructed ileum that a complete division by necrosis of the bowel had occurred, for in no other way could the development of a dead end of ileum be explained.

It was gratifying that after all these vicissitudes the patient was back at work and earning his own living.

Compound Fracture of the Ulna.

DR. G. A. C. DOUGLAS showed a patient with a compound fracture of the ulna involving the elbow joint.

The patient, a male, aged twenty-eight years, had been hit by a car while riding a motor cycle. He had been unconscious and had had a long laceration of the scalp but no evident fracture of the skull. There had been a much swollen right elbow with a punctured wound over the subcutaneous border of the ulna; crepitus had been evident.

The wound of the skin had been cut out and cleaned and then enlarged up and down; the contused tissues had been cut away and the bone exposed. There had been extensive communition of the ulna, breaking it into many fragments at the level of the superior radio-ulnar joint. The upper main fragment had been split into three and there had been a long split running two-thirds of the way down the shaft. Loose fragments of bone had been removed, periosteum carefully saved, the upper fragments had been approximated. The periosteum had been closed as carefully as possible and the wound stitched. The arm had been put up in flexion of a little less than a right angle and supported by a posterior plaster splint from the shoulder to the fingers, the hand being kept supinated. This had been left undisturbed for two weeks, when the plaster had been removed and the stitches taken out, the wound having healed. Towards the end of the third week light massage had been begun and active movement. At the end of five weeks there was flexion, allowing him to touch his shoulder with his fingers and extension to 120°. There was no pain. Pronation was very limited, but was increasing slowly.

Amyloid Nephrosis.

DR. L. J. JARVIS NYE showed a patient who was suffering from anyloid nephrosis. Six months previously he had had a chronic empyema which had drained for a considerable time. On admission to hospital he had had definite œdema of the extremities, ascites and fluid in both pleural cavities. His urine had contained a heavy deposit of albumin with hyaline casts. The systolic blood pressure had been 130 and the diastolic pressure 80 millimetres of mercury. The blood urea had been 38 milligrammes per 100 cubic centimetres and the urea concentration 2·2%. He had been put on high protein diet, as advocated by Epstein, with ammonium chloride and three injections of "Novasurol" had been given. He had responded to these very satisfactorily. His œdema had entirely cleared up and he was anxious to get back to work.

Tuberculosis of the Ileum.

DR. E. S. MEYERS showed a male patient, aged seventy years, who had been born in the Solomon Islands. He had been admitted on May 14, 1929. He had undergone an operation for abscess above the umbilicus thirteen years previously. His wife was alive and well and there was one child who was healthy. On admission he had complained that for the last few months he had had pain in the abdomen, radiating up the chest, dull in character and mostly on the right side. There had been no vomiting. Also he had complained of pain half an hour after meals, this being accompanied by vomiting. Flatulence had troubled him. There had been no loss of weight. The history had been elicited with difficulty. On examination the heart and lungs had manifested nothing abnormal.

The abdomen had moved freely with respiration, there had been no rigidity. Tenderness had been present in the right iliac fossa and in this region a tumour had been felt, kidney shaped, firm, freely movable and slightly tender.

Barium enema had revealed the large intestine well filled to the caecum and with no defects. A mass had been felt below the end of the caecum and had appeared to be extrinsic. Occult blood had been present in the fæces. Urine, examined microscopically, had contained a number of red cells, a few pus cells, but no casts. The blood urea had been 38 milligrammes per one hundred cubic centimetres. The urea concentration figures had been 2·9%, 2·5%, 3%. A blood count had yielded the following information:

Erythrocytes, per cubic millimetre	4,600,000
Hæmoglobin value	90%
Colour index	0·9
Leucocytes, per cubic millimetre	6,400
Neutrophile cells	60%
Lymphocytes	35%
Large mononuclear cells	3%
Eosinophile cells	2%

On May 22, 1929, a normal pyelogram had been obtained and it had been impossible to get the catheter into the right ureter. Indigo-carmine had appeared in the urine in fifteen minutes.

On May 29, 1929, operation had been performed. A lower right rectus incision had been made. The lower end of the ileum had been found to be tuberculous. Adhesions had hidden the appendix and tuberculous glands had been noticed in the mesentery. An ileostomy had been performed and the caecum drawn outside the wound.

Oculo-gyric Crisis.

DR. JOHN BOSTOCK showed a patient with oculo-gyric crisis. He prefaced his remarks by pointing out that spasmodic conjugate movements of the eyes in an upward direction had been known since ancient times. Recently the subject had received prominence on account of its frequency as a sequela in *encephalitis lethargica*. In this connexion it had been pointed out that whilst its occurrence had been taken to presuppose involvement of an eye movement in the region of the nasal nuclei, it was by no means impossibly due to psychological factors following the resultant invalidism. The patient, bowed by his infirmity, figuratively "looked upwards." The case demonstrated at the meeting was hysterical in origin.

M.H., *ætatis* twenty-eight years, a single woman, had been present three years previously at a screen presentation of "The Shiek." She had then noticed that her eyes were affected and she took a "turn." Later whenever she had turned her eyes upwards, these attacks had been apt to occur. The intrusion of a man into the ward almost invariably induced an attack. Although she was not unconscious, she made her limbs rigid and would stare upwards without blinking. The lids moved flutteringly, but the palpebral aperture was never closed; after a while her limbs flexed and she emitted heavy dry sobbing noises. The patient would then get out of bed, apparently not knowing what to do. Although she had previously

repressed the fact, it was elicited at the meeting that she had been subject to sexual interference at the age of seven and eleven. The episode of "The Shiek" had apparently revivified these emotional episodes and had resulted in the hysterical manifestations of the oculo-gyric crisis. As an underlying subconscious motive the wish for sexual excitation undoubtedly existed, apparently unsatisfied in recent years.

Chorea.

Dr. Bostock also showed a man, *ætatis* thirty, who, when admitted, had exhibited choreiform movements. These had improved after tonsillectomy, sedatives and rest. Although the early history was inconclusive, the possibility of adolescent chorea raised the question of a diagnosis of chronic chorea. He pointed out that though chorea tended to spontaneous recovery, a patient showing chronicity raised an interesting speculation as to its frequency.

Diabetes Mellitus and Myxœdema.

DR. CYRIL SHELLSHEAR showed a patient whose *diabetes mellitus* was associated with myxœdema. The patient had first been seen privately, complaining of an abscess of the hand. A routine examination of the urine had revealed the presence of a large amount of sugar, in consequence of which she had been sent to the hospital for further examination. The sugar curve, after the ingestion of glucose had shown a slow rise to the maximum which was maintained; this was common in pituitary dysfunction.

Her symptoms of hypothyreoidism were quite pronounced; her basal metabolism rate was —20%. An attempt to put her on thyreoid extract had been followed by alarming symptoms, and it had been feared that she was going into a comatose condition. This had occurred on two occasions, the second following a dose of 0·03 gramme (half a grain) given three times a day. X ray examination had shown the pituitary fossa to be smaller than normal.

In addition there had been definite ptosis of both eyelids, more pronounced as the day went on. This condition had been very like that seen in *myasthenia gravis*. The condition seemed to be one of general glandular dysfunction.

Infective Arthritis.

Dr. Shellshear's second patient was suffering from arthritis which had not appeared to be due to the gonococcus. However, after seeing a recent article on the use of intravenous injections of "Acriflavine" in gonococcal infections, Dr. Shellshear had decided to try it in this instance as no improvement was taking place. An intravenous injection of five cubic centimetres of a 1% solution had been immediately followed by a drop in the temperature and almost complete relief of symptoms. After the third injection given at three-day intervals, the temperature had become normal and had remained normal.

Secondary Anæmia.

Dr. Shellshear's third patient was suffering from secondary anæmia of unknown origin. The only positive finding was the presence of occult blood in the stools. The gastric juice showed the presence of normal free hydrochloric acid. The Van den Bergh test yielded no reaction. The colour index of the blood varied from 0·6 to 1·0. This patient had first been seen in 1926; the patient had been the first patient in the hospital to be treated with liver extract. On this treatment the patient had done remarkably well. It now nauseated the patient so much that he absolutely refused to take it.

Opinions were asked for as to causation and treatment.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held at the University of Adelaide on September 6, 1929.

Actinomycosis in Sheep.

DR. L. V. BULL gave a short outline of some work on an infective skin lesion of sheep due to an imperfect fungus of the genus *Actinomyces*. In the past the disease had been quite unknown to the majority of sheep owners and where it had been known, only an occasional animal had been found affected. During 1928, however, in two flocks of stud merinos there had been a heavy infection of young ewes, seven to eight months old, and a less severe infection of ewes, one and a half years old. The lesions were scattered over the backs, the sides and the neck and the wool became densely matted together. Usually the areas affected were roughly circular and varied in diameter from about 6·25 to 25 millimetres (a quarter of an inch to an inch). On occasion the skin of the face is also affected.

Specimens of the matted wool and cultures of the *Actinomyces* were exhibited. Attention was drawn to the proteolytic enzyme of the microorganism which readily attacked casein, but under ordinary cultural conditions had little or no effect on other proteins. In the breaking down of the protein ammonia was formed, showing that the enzyme differed from proteolytic enzymes of animal origin.

Testing the Intelligence of Aborigines.

DR. H. K. FRY dealt with the application of intelligence tests to aborigines. By means of using the Müller optical illusion, their ability to measure a line had been estimated. The lines in the illusion figures were also in certain specimens unequal. Only four persons in thirty-nine had given a direct result to the test, the others had had to be gradually worked up to the final adjustment. The illusions in aborigines were as definite as in Europeans. Binet-Simon line tests had been employed, a set of double lines, one of which got progressively longer being used. The illusion effect was the same as in Europeans. With refitting puzzles results poorer than in a four year old European were obtained. The older natives were less capable than the younger, even though the latter had not been educated. A jig-saw puzzle was also demonstrated; it had often been quite reasonably solved. Maze puzzles had also been used and even children of eleven or twelve could do a fourteen year grade maze puzzle.

Using simple line drawing pictures only, natives had made a simple enumeration of objects. In thirty children the illusion of perspective had failed in various ways. A picture of a native dog (dingo) was recognized, while a picture of a native companion was called an emu. A copy of a rock drawing of a dingo was called an alligator in two instances, the rest said "can't know." This drawing was a bizarre one.

Colour sense had been tested with beads. There was no strong selective effort, the native indicating no strong preference. One eleven year old child, quite wild until eight years old and then at school till eleven, was found to be seven years ten months of age when tested on a European code (Binet-Simon). This was considered good in view of the nature of the test.

With the von Frey test the results were very similar to those of Europeans as found by Rivers. Pain sense was identiaal with that of Europeans.

Reaction time measurements were subject to 20% instrumental error. The lowest light response was 0·168 and for sound 0·151. Time lag was shown (twenty seconds) in changing from light to sound (simple dilemma) and finally the time taken was 0·4 second. Snellen's type E for five metres distance had been seen at twelve metres by one, at six metres by many boys and at 9·6 metres by one girl, the average being 1·2 metres. This was seven times the normal. With Snellen's figures the values had been only one half of the previous values; these were more accurate. The E used had not been fully a Snellen type. Tests for intensity of illumination had also been made.

DR. H. C. HOSKING referred to New Guinea natives fresh from the bush who would recognize a conventional drawing for what it was, but could not recognize a photograph of the same. The broken circle test worked very well with the Kanaka, but the female was hopeless. He considered that tests should be based upon facts of native everyday life; in these circumstances they evidenced quite good intelligence.

Infected Wood.

MR. GEOFFREY SAMUEL demonstrated specimens of Czechoslovakian beech which had been infected by a fungus *en route* to Australia. The wood was completely useless for commercial purposes. The infection raised some interesting points with regard to insurance.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held in the University of Adelaide on October 4, 1929.

Scurvy.

PROFESSOR J. B. CLELAND gave an account of some of his experiences as a member of an anthropological expedition to Hermannsburg, Central Australia. At the beginning of 1929 there were at the station of the Australian Inland Mission nearly four hundred natives. Since then about forty had died. A visiting medical practitioner had diagnosed the condition from which the natives had suffered as beri-beri. Those patients seen by Professor Cleland had been suffering from scurvy. The food the natives had been receiving was just sufficient to maintain life. It was therefore possible that beri-beri had occurred previously among the natives. Many of the patients had spongy gums. The condition was evident especially on the palatal side. Bloodstained saliva had poured from the mouth of one small girl when she cried. Several natives had swollen knees. There had been evidence of effusion into the joint cavity; the limbs had been held in a position giving the greatest possible capacity of the knee joint cavity. Contraction of the ham string muscle had been noted in several patients. In some there had been involvement of other large joints. Both recent and old effusions of blood into the muscles had occurred, resulting in indurated areas. In the long standing affections there was œdema of the ankles. Oranges which had been brought by the expedition, had been given to the natives. In three or four days the patients had become brighter and had tried to move about. The gums had healed rapidly. Although improvement had occurred in the majority, one child had died while the expedition was still in Hermannsburg and two had died subsequently, in spite of antiscorbutics. The child who had died, was a boy of twelve who had been ill for two or three months. During that time he had been watched continuously by his parents. They would not permit a *post mortem* examination. Both the natives who had died after the expedition had left, had had œdema of the ankles. Professor Cleland regarded this as an unfavourable sign.

The daily ration of the natives was as follows: flour 454 grammes (one pound), sugar 45·4 grammes (1·6 ounces), rice 30 grammes (about one ounce), barley 15 grammes (about half an ounce), split peas 15 grammes (about half an ounce), meat 45·4 grammes (1·6 ounces). The rations were distributed once a week. The ration was devoid of antiscorbutics and deficient in other vitamins. Despite this, the native with relatively few exceptions appeared to be in excellent condition. The bush natives lived largely on lizards, grubs, wallabies, seeds of various kinds and *Calandrina balonnensis* and *Portulaca oleracea*, vegetables presumably endowed with antiscorbutic properties. Owing to the five year drought neither of the vegetables could be obtained at the time of the visit. A bush family of man, wife and three children who had arrived at the mission station at the time, had been in a starved condition.

Professor Cleland appended an account of the occurrence of scurvy among the early explorers and colonists of Australia. A classical description had been published in Lord Anson's "Voyage Around the World." He also referred to blood grouping. It has been found that two-thirds of the aborigines at the station of the Australian Inland Mission at Hermannsburg belonged to Group A and one-third to Group O. Professor Cleland used von Dungern and Hirschfeld's annotation instead of the more usual classification of Moss.

A discussion on deficiency diseases and vitamins followed.

Congress Notes.

INTERNATIONAL PHYSIOLOGICAL CONGRESS.

THE thirteenth International Physiological Congress was held at Harvard University, Boston, in August, 1929. In an address of welcome to the members Professor William H. Howell pointed out that within fifty years physiology had begun its career in the United States as an independent science with special facilities for instruction and research and the training of its own group of workers. The real birth of physiology lay in the establishment of the laboratories of Bowditch at Boston, of Newell Martin at Baltimore and of Chittenden at New Haven. Into these laboratories were transplanted from Europe the spirit and methods of Ludwig and Bernard, Michael Foster and Kühne. From these modest beginnings had sprung the splendid laboratories of physiology and medicine, with hundreds of workers and thousands of contributions to the advancement of knowledge.

The opening address was delivered by Professor August Krogh, of Copenhagen, on the progress of physiology. He stated that almost every worker in physiology had given some thought to the general progress of the science and to the problems raised by its growth. His aim would be to draw attention to these problems in the hope that means might be found to solve them. He had tried to count from the abstracting and index journals the number of papers published upon physiological subjects. In 1901 3,800 papers had been abstracted and during the first decade of the century only minor fluctuations in this number had taken place. In 1912 the figure had been 6,700. In 1920 11,500 papers had been dealt with and in 1926 the figure had risen to 18,000. He had the impression that physiological papers were on an average shorter than they had been twenty-five years previously, but a sample counting had failed to substantiate this impression and showed the average length to be still about ten pages. It was interesting to note that in 1901 only 2·5% of articles had been published in America or by American authors, while in 1926 nearly 20% of the total consisted of papers published in American journals of physiology.

Physiology had been brought into much closer contact with the allied and fundamental sciences. Twenty years previously the application of statistical and mathematical analysis of problems had been uncommon and unfamiliar to physiologists and some had even deprecated it. There was still room for further progress, but statistical weapons were wielded successfully by a large and increasing number of investigators. In many ways results of researches in pure chemistry and physics were adapted to their problems and he trusted that they could claim some reciprocity, since the use of micro-methods, initiated mainly within their own domain, was spreading to other provinces of science. It was extremely gratifying to note how even the most advanced ideas and methods of physics and chemistry were inspiring the work of a few of their colleagues.

While they had every reason to rejoice over the growth and progress of their science, he thought that they must admit that there was room for some improvement and that some aspects of the situation were unsatisfactory. Physiology was growing unwieldy. It was impossible for any single human being to be familiar with modern physiology in all its branches in the sense in which great teachers of twenty or more years before had been familiar with the physiology of their time. They had to face the division of physiology into more or less separate sciences. They were confronted with the problem of finding right lines of cleavage. Research was bound up closely with teaching and the lines of cleavage would be determined by the university chairs and departments which were brought into existence. It was the practical teaching situation which had made pharmacology an independent science. They had seen biochemistry obtain independent chairs in laboratories in the universities of many countries and they all agreed that this had been a most happy development which would be carried on into those universities

and medical schools which had not yet effected the separation.

A few universities, and Copenhagen among them, had made a new departure in creating separate chairs and departments for biophysics. This was justified from the point of view of teaching and for other practical reasons. Their physical problems had been usually rather elementary, but he would admit that the situation was rapidly changing and it was quite possible that new discoveries like that of radiation from rapidly growing tissues, might make biophysical departments generally desirable or even necessary.

Physiology as a science has taken its origin from the necessities of practical medicine and the large majority of workers in physiology had the benefit of a medical education and held their appointments in medical schools. Nevertheless, physiology was an independent science; much of the work done in physiological laboratories had no direct relation to medicine. The line of development which he thought should be followed, was to establish a branch of physiology which was much more intimately in contact with practical medicine, and a branch which was much more independent. He thought work done on the American side of the Atlantic Ocean had contributed more than anything else to the understanding of the fact that the problems of disease were mainly physiological and could not be solved by the methods of clinical observation, morbid anatomy, bacteriology or serology alone, even when these sciences were essential. It was the functional reaction of the organism to the attack of disease and to therapeutic measures which was after all the central problem. A large and increasing proportion of physiological research was carried on in hospital laboratories by men and women who were at the same time practising the art of healing. Much of this work was very valuable, but frequently the outlook was rather narrow and he believed that a great deal could be gained by expert physiological direction and coordination. He thought that the time had come when special chairs and laboratories should be established for the physiology of disease, morbid physiology or experimental medicine, if they prefer that name. The main point was that the leaders of such laboratories should have no regular duties connected with the treatment of patients, but they should have a small number of beds at their disposal for the temporary study of selected conditions and they should be in close and constant contact with the clinical wards. He had learned that the theoretical problems regarding kidney function required for their solution a close study of clinical manifestations and he could not doubt for a moment that great benefit to patients would ultimately result from such a study. The other line of development which he would suggest, was not such a pressing need, but would ultimately, he believed, be of scarcely less importance. It was the creation of chairs and laboratories for comparative physiology, animal physiology or zoophysiology in the science schools in close cooperation with the department of zoology. The name did not matter much. He wished to say a word for the study of comparative physiology for its own sake; they would find in the lower animals mechanisms and adaptations of exquisite beauty and the most surprising character; he thought that nothing could be more fascinating than the sense and instincts of insects as revealed by modern investigations. A good beginning had been made in the study of this group of problems and an increasing number of papers was published which could be classed as comparative or zoophysiological.

When he attempted to pass in review the physiological literature of the day he noticed certain defects which were too common and could no doubt be remedied. In a recent small book of instructions for medical writers he found a statement that what were needed in scientific problems were facts, more facts and still more facts. He ventured to disagree emphatically with that statement. Facts were necessary, of course, but unless fertilized by ideas, correlated with other facts and illuminated by thought, he considered them as material only. He was prepared to submit the thesis that too many experiments and observations were being made and published, while too little thought was bestowed upon them. The statement was made too frequently in physiological papers that a certain experiment had been repeated on, say, forty-seven animals. Often, though not always, such a routine procedure was sheer waste of time and animals. At the root of the apparent diligence lay a mental inertia which carried the experimenter along the accustomed groove with a minimum of exertion of the mind. Physiological experiment required constant exertion and constant attention to details which might be trivial, but might turn out to be of vital importance. It was an almost invariable custom for editors of journals to reject papers which did not contain new facts. It was natural to be sceptical towards reasoning not supported by facts, but it must happen to many physiologists that their thoughts were concerned with facts which were incompletely understood by those who had brought them forward. On more than one occasion experiments had been published for which much more consistent explanations could be offered than had been adopted by their authors. A more liberal interpretation of the rules of the journals would serve the interest of their science. He ventured to say that the publication of papers in which facts already known were discussed, should be encouraged.

Physiology must be carried on as someone had aptly said, as a guerilla warfare against the unknown by quite small units. There was no need for an extensive organization of research, but there was much need for voluntary cooperation on a limited scale between individuals and laboratories. There were many problems which could be successfully attacked only if experimental physiologists cooperated with histologists, with chemists or physicists or with clinicians; some problems would require the combined efforts of several of these groups.

There was a wide and fruitful field for organization in the services necessary to the physiologist. He had no doubt that the methods of indexing, abstracting and reviewing physiological literature could be improved, so as to give better service at less cost. It was most desirable that one system should be agreed upon and used by all the leading physiological journals and department libraries throughout the world. It was quite conceivable that even an inferior system universally adopted would be much better than any system in use locally or a general lack of system. It was, he thought, a wasteful procedure that the same paper should be independently recorded and abstracted in a great number of different abstracting journals. He trusted that the congress would give some attention to these matters.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 102, 106 and 110 of October 31, November 21 and 28, 1929.

AUSTRALIAN MILITARY FORCES.

First Military District.

Australian Army Medical Corps.

The provisional appointment from the Australian Army Medical Corps Reserve of Captain V. F. O'Neill is terminated, 10th November, 1929. *To be Captain (provisionally)*—Honorary Captain V. F. O'Neill, from the Australian Army Medical Corps Reserve, 11th November, 1929.

To be Majors—Captains M. L. D. McKeon and R. E. Douglas, 19th August, 1929, and 1st September, 1929, respectively.

Australian Army Medical Corps Reserve.

The resignation of Honorary Captain J. Brown of his commission is accepted, 14th October, 1929. Captain J. A. Goldsmid and Honorary Captain H. A. Legge-Willis are retired, 4th November, 1929, and 2nd November, 1929, respectively.

Unattached List.

To be Honorary Captain—Norman Leslie Sherwood, 25th September, 1929.

Second Military District.

Second Division: Staff.

Lieutenant-Colonel R. A. P. Waugh, V.D., Australian Army Medical Corps, is appointed Assistant Director of Medical Services and is granted the temporary rank of Colonel whilst holding such appointment, 9th October, 1929.

District Base: Staff.

Lieutenant-Colonel R. W. W. Walsh, D.S.O., Australian Army Medical Corps, is appointed Assistant Director of Hygiene, 1st September, 1929, *vice* Major H. Sutton, O.B.E., who relinquishes the appointment on 31st August, 1929.

Australian Army Medical Corps.

To be Lieutenant-Colonel—Major T. M. Furber, 1st September, 1929. *To be Majors*—Captains S. U. Gentile, J. C. Shand and T. Hamilton, 1st September, 1929. Lieutenant-Colonel R. A. P. Waugh, V.D., is transferred from the Unattached List with regimental seniority next after Colonel J. C. Storey, O.B.E., V.D., 9th October, 1929. Lieutenant-Colonel R. W. W. Walsh, D.S.O., relinquishes the command of the 5th Field Ambulance, 31st August, 1929. Major H. Sutton, O.B.E., and Captain R. B. Austin are transferred to the Unattached List, 1st September, 1929, and 1st July, 1929, respectively. Major C. C. Corlis, M.C., and Captain J. T. Paton are transferred to the Australian Army Medical Corps Reserve, 1st July, 1929. Captain J. C. Belisario is supernumerary to the establishment pending absorption, 1st July, 1929.

Australian Army Medical Corps Reserve.

Major G. L. Kerr is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 3rd November, 1929. Honorary Captains A. E. Gibbes and E. W. Buckley are retired, 3rd November, 1929, and 12th November, 1929, respectively.

Award of the Colonial Auxiliary Forces Officers' Decoration.

Australian Army Medical Corps.—Lieutenant-Colonel W. R. C. Beeston.

Third Military District.

Australian Army Medical Corps.

To be Lieutenant-Colonels—Majors R. C. Withington and W. W. S. Johnston, D.S.O., M.C., 1st August, 1929. *To be Captain (provisionally) supernumerary to the establishment pending absorption.*—Geoffrie Carthew Burston, 12th October, 1929.

Captain J. F. Patrick is transferred to the Unattached List, 1st July, 1929.

Australian Army Medical Corps Reserve.

Honorary Captains G. Gocher and G. A. Hagenauer are retired, 24th October, 1929, and 3rd November, 1929, respectively.

Fifth Military District.

Australian Army Medical Corps.

To be Major—Captain L. E. Le Souef, 24th September, 1929. The provisional appointment of Captain A. R. Home is confirmed.

Captain C. W. Harris is transferred to the Australian Army Medical Corps Reserve, 25th October, 1929.

Sixth Military District.

Australian Army Medical Corps Reserve.

Major P. A. Maplestone, D.S.O., is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 1st October, 1929.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on December 2, 1929.

The degree of Master of Surgery (Ch.M.) was conferred *in absentia* on Stanley Augustine McDonnell, M.B.

The following appointments were approved:

Mr. J. R. Jones, B.V.Sc., as Lecturer in Veterinary *Materia Medica*, Therapeutics and Pharmacy in the School of Veterinary Science.

Dr. F. W. A. Clements as Acting Radium Registrar at the Radium Clinic at the Royal Prince Alfred Hospital.

The Agent-General for New South Wales advised that Dr. Whitridge Davies had accepted the Chair of Physiology and would arrive in Sydney on 28th February next.

Special Correspondence.

CANADA LETTER.

BY OUR SPECIAL CORRESPONDENT.

Recent Congresses.

THE months of May and June with the long days, sunshine and balmy air make a splendid environment for both the auspicious services associated with the conferring of medical degrees on the young graduates and the assembling of the medical associations to take stock of the work accomplished during the past year.

The Ontario Medical Association held its annual meeting in Hamilton. The attendance was large and the papers and discussions attracted attention as they dealt with the problems met with in everyday practice, such as sinus infections, jaundice, arthritis and hernia. Dr. Charles Mayo gave an interesting talk, spiced as usual with many humorous skits, on the functions of the autonomic system.

Dr. John Ferguson, one of Canada's Nestors of medicine, was chosen president for next year. He has rendered Canadian medicine, the Toronto Western Hospital and the library of the Toronto Academy of Medicine services of inestimable value. He combines very effectively culture, experience, industry and dauntless courage with constructive acumen. His genius and optimism were the paramount factors in founding the Toronto Western Hospital and in transforming a small one-roomed free dispensary into a palatial block of building with large grounds and central location. Recently this hospital received a munificient donation from two wealthy citizens, Mr. Loblaw and Mr. Cork, of a million dollars, to be supplemented by two millions more from other sources, to construct on another site a main building with the present one as a branch.

The Canadian Medical Association held its annual meeting in Montreal and was largely attended. Several distinguished men from Great Britain, France and the United States were present. Dr. Harold Smith, of Winnipeg, was chosen President for next year. Dr. Smith has also been elected President of the British Medical Association and will preside over the joint meeting next summer in Winnipeg. This great meeting will be a most notable one. It will bring together a host not only of Great Britain's most distinguished physicians and surgeons, but many from her dominions, as well as from the United States, France and other countries.

Winnipeg, the great metropolis of our middle north-west, can furnish ample accommodation for all who may come, while the welcome and generosity of her citizens will be warm and great enough to withstand any strain.

The vacancy caused by the untimely and lamentable death of Clarence Starr, Chief of the Surgical Department of the University of Toronto and of the Toronto General Hospital, has been filled by the appointment of Dr. Gallie. The new chief is in middle life, has rendered brilliant service in the Sick Children's Hospital and has won dis-

tinction as a surgeon. He enjoys in a remarkable degree the confidence of the medical profession.

Infantile Paralysis.

An epidemic of infantile paralysis (acute anterior poliomyelitis) began in midsummer in one of the Western Provinces of Canada, and spread rapidly eastward. State medicine was immediately mobilized, circulars were sent broadcast informing the laity of the symptoms and character of the disease and the need of prompt medical supervision. An abundant supply of convalescent serum was provided. The epidemic was brought under control quickly and judging from press reports (official report not at hand), interviews of ministers of health, health officers, and of physicians, the convalescent serum furnished a most effective remedy against this disease, since in only 2% or 3% of the patients did paralysis follow. The wide diffusion, through press and circular, of knowledge pertaining to the disease, prompt medical attention, a supply of convalescent serum and the mobilization of state medicine proved to be most effective measures, in not only stamping out the epidemic, but in preventing the dire consequences that only too often follow the destructive effects of the disease on the motor cells.

Physiotherapy Course.

A new long needed and a most valuable addition has just been made to the medical curriculum of the University of Toronto, namely the establishment of a full course in physiotherapy. The greatest of all medical problems is the relief of suffering, cure and prevention of disease and from the "most ancient of days" physiotherapy has occupied a great place not only in the preservation of life, but also in the mitigation of suffering. However, in the rapid accumulation of knowledge pertaining to biology, anatomy, physiology, bacteriology, interest in physiotherapy waned until finally it ceased to be taught in our medical colleges. Recently there has been a *renaissance* in physiotherapy and a special course is now provided in many of our universities.

Two problems are involved in physiotherapy, the physicians and the technicians. Before physiotherapy can be intelligently and efficiently used, the physician must acquire at least as much knowledge concerning its potentialities as those of drugs. His prescription concerning the use of physiotherapy must be just as intelligible to the technician as the one given to the druggist. It is just as absurd to send a patient to the technician as it would be to send him to the druggist, without a prescription. Hereafter no student should be granted a degree without knowing as much about the use of physiothearpy, as he knows about the use of drugs. The technician must acquire the art of applying intelligently the technique used in physiotherapy, but always under instruction of the physician.

Current Events.

The general prosperity throughout Canada is reflected in all the departments of our universities, by the large number of students who have registered. The fair sex is the most numerous. A few years ago, women in early mid-life were the only ones to be seen, but now the great majority seems at least to be only in the "teen-age." Formerly the female of the "student species" dressed in rather sombre hues, but now the latest model on the fashion plate is everywhere in evidence.

Post-graduate work is being provided more and more each year. Special courses are being given at several of the hospitals, and members of our hospital staff have gone to different parts of the Dominion to deliver addresses.

There is a provincial election on, and quite a large number of physicians is seeking seats in the legislature. The control of liquor by government permits is the "bone of contention." Two and a half years ago one of our political parties made its appeal to the electors on "Government control of liquor" and a large number of the leading physicians adopted it and their names with all their degrees appeared daily in the press. Not a single name appears in this election in favour of "Government control."

Physicians meet to-morrow (October 20, 1929) as it is set apart as St. Luke's Day, at the Toronto Academy of Medicine to attend the religious services in Wycliffe College.

Correspondence.

TONSILLECTOMY.

SIR: Dr. Hornabrook's letter in your issue of today shows either an alarming ignorance of the advances in surgery of the tonsils or a desire to pose as a reformer and play down to the gallery.

If he knew the conditions and results of the operation as generally performed "by an operator who knows his work," blindly working with a blunt guillotine under "Kelene" or repeated doses of ether with the consequent loss of blood, and the present day operation by dissection which is slower and safe, I think he would "for ever hold his peace."

All surgeons attached to hospitals have seen the tragic results of the efforts of the "operator *et cetera*" to remove tonsils with the guillotine.

The old surgeons cut for stone, operating very quickly, with but few instruments and little or no anæsthesia. Would Dr. Hornabrook suggest a return to this condition in preference to suprapubic cystostomy?

All modern operations involving skill are usually criticized by the surgeons who do not perform them, the excuse being the public good, whereas the real reason is: "I cannot do it, therefore I don't believe in it."

The remark about the "pantechnicon" displays a profound ignorance of the advance in anæsthetics and calls for no comment.

Yours, etc.,

CHARLES F. WARREN,
Surgeon, Ear, Nose and Throat Department,
Sydney Hospital.

November 29, 1929.

SIR: Kindly allow me space to congratulate Dr. Hornabrook for his protest against the operation of tonsillectomy as performed by many specialists today. It is long overdue.

I have seen many of these dissection operations performed and if one is to judge by the machinery of the anæsthetist around the operating table and the awesome instruments designed to keep the mouth opened and the tongue depressed and the assistant endeavouring to thrust some form of sucking apparatus into the already overcrowded mouth, the only conclusion to arrive at is that what should be a simple operation is made difficult and at times positively dangerous.

Is all this fair to the patient? Surely not. With all the manipulation there is an increased risk of secondary hæmorrhage and a longer convalescence with its added hospital expense. Then in addition to the specialist's fee there is the anæsthetist to be paid. As he too is said to be skilled, his fees are in proportion.

Why, then, should a cumbersome and prolonged operative procedure result out of what used to be and still is in skilled hands a simple operation? It seems to be a retrograde step in the specialty of ear, nose and throat. Everybody knows or should know that the guillotine is applicable to the great majority of cases at any age. This operation offers to the skilled technician a simple and short method of enucleating tonsils and as it entails less discomfort, less risk and a shorter convalescence and of necessity less expense to the patient, it should commend itself for general use.

Let the dissection methods be reserved for special cases, for example, tonsils that have been the seat of repeated attacks of quinsy and septic tonsillar remains and then an efficiently employed local anæsthetic affords a simple method of doing the operation.

I think it is time that some of the modern tonsil experts examined their conscience.

Yours, etc.,

"PRACTITIONER."

December 3, 1929.

STEINACH'S OPERATION.

SIR: Dr. Paul Dane asks through your columns for first hand information concerning the after results of the Steinach operation.

About three or four years ago I performed this operation three times at Saint Joseph's Hospital.

Case I. manual worker, *ætatis* nineteen. *Paralysis agitans.* A classical case. Three months after operation patient felt better, gained weight, tremor improved. Present condition, pitiful state of advanced *paralysis agitans.*

Case II. Male, *ætatis* thirty-seven, sedentary worker, childless and desirous of children. Sexual vigour poor. Premature ejaculations. After operation noted improvement which may have been psychological. Present condition, same as before operation.

Case III. Working man who had had *encephalitis lethargica, ætatis* thirty-five. Condition, premature senility. Walked, talked and acted like a man of eighty years. (With apologies to vigorous old gentlemen of eighty years.) Painfully slow to comprehend conversation. Three months after operation felt better, looked better; gained one stone mass (in spite of social condition which following the encephalitis is one of poverty). Seven months after operation had improved to the extent of feeling fit to look for a light job. I saw him eighteen months ago. His condition was just as bad, if not worse than before operation.

The three operations were done according to Peter Schmidt's description, the artery to the *vas* being preserved. Each was monolateral. A local anæsthetic ("Apothesine") was used.

One swallow does not make a summer and three Steinach operations do not make a Faustian spring; nevertheless one thinks that the writers who advocate the operation, are filled with an enthusiasm not generally shared nowadays by the profession.

Dr. Paul Dane will get a fairer idea of the yearly swing of the pendulum for and against this operation if he refers to the "Medical Annual," 1925 to 1927 inclusive.

Yours, etc.,

KEVIN BYRNE.

Lakemba,
New South Wales.
November 29, 1929.

DUODENAL STENOSIS.

SIR: I was very much interested in the report by Dr. Buchanan of a case of duodenal stenosis appearing in your issue of November 2, owing to a recent similar experience occurring in my own practice. On October 15 last I confined a woman for her sixth child. The confinement was normal except for a rather excessive amount of *liquor amnii*, and the child, a male, appeared normal. However, twelve hours after birth he vomited a copious amount of bile-stained, frothy fluid and this was repeated in six hours' time and subsequently after every feed. I examined the child when twenty-four hours old and found the following abnormalities: a systolic murmur heard at all heart areas with no cyanosis and a distension of the upper half of the abdomen with visible peristalsis in the stomach. No tumour was palpable and no meconium or any action of bowel had been passed. Owing to the presence of what appeared to be bile in vomit and the lack of tumour, a diagnosis of congenital occlusion high up in the small bowel was made. The infant rapidly got worse and became cyanosed, ultimately dying when sixty hours old. A *post mortem* examination was made, the brain not being examined. The heart was found to have a patent interventricular septum and this was the only lesion present in the heart. In the alimentary canal a condition very similar to that in Dr. Buchanan's case was found. The stomach and the first part of the duodenum were grossly dilated, the pylorus atonic, easily admitting a finger. At a point just below the ampulla of Vater there was a gap in the continuity of the bowel, the two ends being joined together by a fine fibrous cord about one-half inch long. The rest of the small intestine and the large bowel were collapsed.

Yours, etc.,

L. R. MALLEN.

Riverton, South Australia.
November 5, 1929.

PROLAPSUS UTERI.

SIR: I have already expressed myself in agreement with Dr. H. C. E. Donovan as to the value of ventro-suspension in certain cases of uterine prolapse and I appreciate the fact that he has "the greatest respect for the work and opinions of the late Arthur Nyulasy." Victor Bonney, it may be added, has similarly expressed himself.

But while Dr. Donovan candidly admits that his comparison of "looping the cardinal ligaments" with Wertheim's operation "is an exaggeration," is it quite fair to exaggerate anything in a scientific discussion?

Yours, etc.,

FRANK A. NYULASY.

Melbourne.
November 23, 1929.

TÆNIA SAGINATA.

SIR: It may be of interest to report the following case of above tape worm.

F.M., *ætatis* twenty, Englishman, lived in Australia for the last nine years, consulted me for pain in the epigastrium of four weeks' duration. As the pain for the last two weeks had been coming on at 1 a.m. and kept him awake for about three hours each night, he had sought treatment. Dieting and salines relieved this pain, but four days afterwards he passed three inches of tape worm. Preliminary starvation, purgation and one and a half drachms of *Felix-mas* resulted in his passing a large mass of white tape worm. This was taken to Melbourne University and identified as *Tænia saginata*, the cystic form of which occurs in "measly beef," especially in the tongue and muscles of mastication. The head was not found. The patient feels very well, but will probably require a further vermicide later on. History elicited the fact that he eats a lot of underdone beef. I was informed at the university that this tænia was of rare occurrence in Australia.

Yours, etc.,

J. SCOTT PATON.

609, Lygon Street,
North Carlton, Victoria.
November 25, 1929.

TESTIMONIAL TO MR. W. FILMER.

A VERY old identity at the Melbourne Hospital, W. Filmer, will complete fifty years of service in that institution on January 21, 1930. He has given demonstrations of surgical instruments to all students who have passed through the hospital during this time. Mr. Filmer is remembered with affection by all Melbourne graduates and the honorary medical staff of the hospital believes that his jubilee as a servant of the Melbourne Hospital should be recognized by a testimonial. Any old Melbourne Hospital student who wishes to subscribe to the fund which is being collected for this purpose, is requested to send a cheque to the Medical Superintendent of the Melbourne Hospital as soon as possible.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of *The Medical Act* 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Bray, Sylvia Dean, M.B., B.S., 1929 (Univ. Sydney), Sydney Hospital.

Julius, Solomon, M.B., 1929 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Skelton, Esther Isobel, M.B., B.S., 1929 (Univ. Melbourne), Mildura.

For additional registration:

Evans, Wilfred, M.R.C.P. (London), 1928.

Books Received.

DISEASES OF THE CHEST AND THE PRINCIPLES OF PHYSICAL DIAGNOSIS, by G. W. Norris, A.B., M.D., and H. R. M. Landis, A.B., M.D., Sc.D.; with a chapter on the Transmission of Sounds through the Chest by C. M. Montgomery, M.D., and a chapter on the Electrocardiograph in Heart Disease, by E. B. Krumbhaar, Ph.D., M.D.; Fourth Edition, revised; 1929. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Royal 8vo., pp. 954, with illustrations. Price: 50s. net.

MEDICAL AND D.P.H. EXAMINATION PAPERS: 1926-1929; 1929. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 186. Price: 3s. 6d. net.

THE EXTRA PHARMACOPŒIA OF MARTINDALE AND WESTCOTT, Revised by W. Harrison Martindale, Ph.D., Ph.Ch., F.C.S.P. Nineteenth Edition; Volume II; 1929. London: H. K. Lewis and Company, Limited. Foolscap 8vo., pp. 792. Price: 22s. 6d. net.

A MANUAL OF MIDWIFERY FOR STUDENTS AND PRACTITIONERS, by Henry Jellett, B.A., M.D. (Dublin), F.R.C.P.I., L.M., and David G. Madill, B.A., M.B., B.Ch., B.A.O. (Dublin), L.M.; Fourth Edition; 1929. London: Baillière, Tindall and Cox. Royal 8vo., pp. 1293, with illustrations. Price: 25s. net.

Diary for the Month.

DEC. 13.—Queensland Branch, B.M.A.: Branch (Annual).
DEC. 17.—Tasmanian Branch, B.M.A.: Council.
DEC. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. Reginald Francis Matters (B.M.A.) has been appointed Temporary Honorary Clinical Physiologist, Adelaide Hospital, Adelaide.

* * * *

Dr. Alan Frank Hobbs (B.M.A.) has been appointed Temporary Honorary Assistant Pathologist, Adelaide Hospital, Adelaide.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page XX.

PEAK DOWNS HOSPITAL, CLERMONT, QUEENSLAND: Resident Medical Officer.

THE QUEEN'S (MATERNITY) HOME, ROSE PARK, SOUTH AUSTRALIA: Resident House Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.